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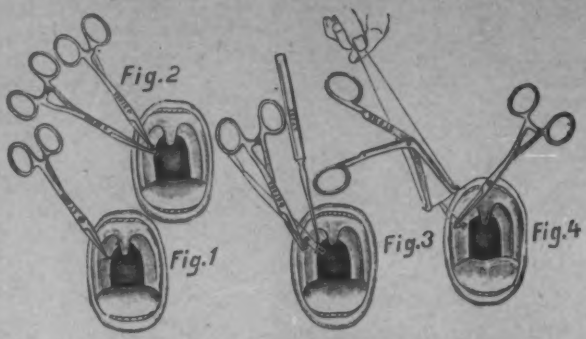

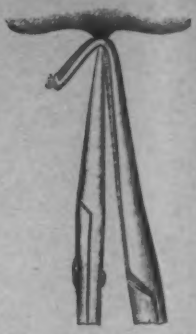

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THE
LARYNGOSCOPE.

VOL. XXXVIII APRIL, 1928. No. 4

ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

AN APPROACH TO THE PROBLEM OF PAIN FIELDS, WITH SPECIAL REFERENCE TO THOSE ASSOCIATED WITH DISEASES OF THE NOSE AND THROAT.*

DR. F. H. PIKE, New York City.

The term ganglion, applied to any structure lying well outside of the central nervous system, immediately brings to one's mind the idea of the sympathetic nervous system with its peripheral nerve cells and fibres; and the term sphenopalatine ganglion calls up the picture of the sympathetic nerves of the head. But only a short period of meditation was necessary to show that a consideration of the cranial sympathetic nerves would not carry us very far in an understanding of the symptoms observed in those diseases of the nose in which the sphenopalatine ganglion is affected. I crave your leave, therefore, to depart from the limits imposed by the title printed in the program and to wander in such other paths as now seem open to me in the following remarks. For the symptoms described in connection with disease of the sphenopalatine ganglion present a complexity and variety which appear to demand more than one basis of explanation.

Without enumerating the symptoms of diseased conditions of the sphenopalatine ganglion or of its connections in detail, they appear to me to fall into three general groups:

1. Pain in the peripheral areas supplied by the branches of the fifth cranial, and possibly of the seventh cranial, nerve which pass

*Read before the Section on Laryngology and Rhinology, New York Acad. of Medicine, Feb. 3, 1927.

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by the ganglion or through other diseased regions of the nose and the sinuses. To this pain should be added the glandular effects due to stimulation of the autonomic or other secretory fibres passing through or arising in the ganglion.

2. Pain in regions remote from any area innervated by the fifth or seventh cranial nerves. The sensory distributions of some branches of the upper cervical nerves are cases in point.

3. Symptoms appearing in the cardiovascular, gastrointestinal and genital systems. Of these I will have little to say here. One may mention in passing that Herbert Mayo was perhaps too optimistic when he said that the second cranial nerve was the first one whose reflex effects gave rise to any difficulty for the physiologist. I am perfectly willing to admit that the suppression of menstruation which sometimes follows the application of an argyrol pack in the nose presents certain difficulties which I am not at present able to explain. Whether the afferent path from the nose lies through the first, the fifth or the seventh cranial nerve, or through the autonomic nerve supply is unknown at present.

The explanation of the first group of symptoms appears reasonably clear. It is a cardinal principle of physiology that a nerve may be stimulated at any part of its course. In the case of a somatic sensory fibre, the sensation is referred to the peripheral distribution of that fibre. Pain in the knee from irritation of the trunk of the obturator nerve in tuberculosis of the hip joint is a familiar clinical illustration. The tingling of the fingers when the ulnar nerve is struck against a sharp corner is familiar to all. That inflammatory processes involving a sensory nerve trunk may give rise to pain seems equally clear. Whether or not the branches of the fifth and seventh cranial nerves actually pass through the ganglion or merely go by the sphenopalatine ganglion does not appear so significant for the explanation of the pain. The fact remains that these nerve trunks lie in the diseased region. Head's figures showing an area of pain in the region of the eyes in disease of the sinuses is understandable on this basis. In passing, I may remark that the problem of the morphological constitution of the sphenopalatine ganglion seems open to experimental attack.

The explanation of the second group of symptoms demands a more extended search. Head shows an area of pain at the back of the head and upper part of the neck as symptomatic of sinus trouble or diseased teeth. It is this pain, remote from the diseased region or from the peripheral distribution of any of the nerves passing

through it, which is puzzling. From the time Mackenzie first held the afferent fibres of the phrenic nerve—and it clearly has afferent fibres—responsible for the pain in the region of the right shoulder in some cases of disease of the liver, up to the present, we have heard much about referred pain. Involving, as it did and still does, much of mystery with reference to the conduction of nerve impulses, it seemed a good heading under which to group many things whose explanation was otherwise unknown. Names save much thinking, and having found a name for these puzzling phenomena, we forthwith ceased to think about them as we should. The diagnostic significance of pain, however, seems no less today than when Hilton published his surgical classic bearing that title. But does the hypothesis of referred pain agree with all the facts as we know them today? Without denying the validity of the hypothesis of referred pain as a whole, I feel strongly that there are some cases commonly included under referred pain which do not properly come in that category. The beginnings of my skepticism date back 15 years or more, and I may perhaps make myself a little more intelligible by recounting some of the incidents along the route of growing doubt.

The first case that made me thoughtful was one of intense pain, uncontrollable by narcotics, in the lower lumbar region. There was some degeneration of the muscles in the gluteal region, indicating some process of considerable gravity in the nerves, and the pain was referred to the peripheral regions innervated by nerves passing out through the intervertebral foramina of the lower lumbar region. The patient, a woman of about 45 years, had a positive Wassermann, a positive von Pirquet, and gave a history of hysterectomy for carcinoma of the cervix some years earlier. Neither syphilitic neuritis nor tuberculosis of the spine seemed a probable diagnosis. The known relations of the pelvic lymphatics to the nerves affected and the extent of degeneration of the muscles, incompatible, as it seemed to the friend who looked over the case with me, with the slight involvement of the vertebrae themselves, indicated a metastatic growth of the tumor about the nerve roots. The operation for the relief of pain-section of the dorsal roots of the lumbar nerves affected, was the first of its kind ever done by the surgeon to whom the case was referred. No tubercular involvement of the spine was found at operation. Such things have grown more familiar to medical men since that time, but they were not so well known then. The moral of this case is that we were not dealing with a case of referred pain from pelvic disease, but with a pain arising from a disease process involving the trunks of the nerves themselves. Other cases of

intense pain in the back arising from metastases of tumors of the prostate have come to my notice since that time.

A few years later another puzzling case came to my notice. The patient—a young woman—had severe and continuous pain back of the right ear. Closer observation showed that the area in which the pain was localized corresponded closely with the peripheral distribution of the small occipital nerve—arising from the second or third cervical nerve roots. The restricted area of pain seemed to preclude any general process in or about the vertebrae and involving the nerve roots themselves. An otologist and an X-ray plate confirmed my opinion that we were not dealing with a mastoid abscess. Nasal sinuses and tonsils seemed equally blameless. X-ray showed an abscess at the root of an upper premolar on the right side, but the tooth itself was not painful. The pain back of the right ear subsided when the abscess was drained.

How is one to explain the pain in the peripheral distribution of the small occipital nerve? The idea of referred pain did not impress me at the time and it appeals to me still less now. It requires a greater stretch of the imagination than I am capable of withstanding to suppose that an abscess, not painful in itself, could excite some afferent but nonsensory nerve in such a way as to set up a disturbance in a remote peripheral nerve with which it had no known anatomical connections. I am not ready to admit that a nerve impulse may jump a gap from one nerve trunk to another lying at some distance from it. As a physiologist, I would say that such a jump was impossible. Fortunately, none of these suppositions seem necessary to account for the fact of the pain in the peripheral distribution of the small occipital nerve. For, if one follows the lymphatic drainage from the upper molar teeth, one is finally led to a group of lymphatic glands lying right by the small occipital nerve as it emerges from the deeper structures of the neck and turns upward to pursue its course back of the ear. In the case in question these glands were enlarged and plainly palpable. My present view is that a sufficient process was set up in these lymphatic glands to cause irritation of the small occipital nerve in their immediate vicinity.

The problem which the student of the physiology of the nervous system meets in this connection may be termed the problem of pain fields. Head recognized the fact that there were areas of pain associated with disease processes in regions at some distance from the area of pain. His figures may be taken as a preliminary and, in some instances as a detailed, statement of the problem. Still earlier than Head, Hilton[1] had studied the relations between the distribution

of peripheral nerves and the occurrence of pain in particular regions. An illustration taken from another field of medicine may help in making the somewhat vague conception of pain fields a little clearer. A surgical friend who does much operating for the removal of malignant tumors once mentioned the problem of cancer fields. He had in mind, as nearly as I can grasp the idea, not only the site of the malignant growth which was to be removed, but the whole field of lymphatic drainage from the original site of the tumor, or any other possible channels along which metastases might occur. Similarly, in the consideration of a pain field, we must have in mind, not only the peripheral nerve field to which the pain is referred, but also any processes of whatsoever nature and wheresoever originating, along the course of that nerve which may give rise to the pain. Perhaps one could deduce the title of this paper from these general statements, and show the idea in mind in calling it an approach to the problem of pain fields.

But can we invoke swollen lymphatic glands and the processes set up in them as an explanation of the pain in the muscles of the back of the neck and even in the shoulder in cases of disease of the sinuses or other regions about the sphenopalatine ganglion? I believe that we can.

If one follows out the field of lymphatic drainage from the sinuses and other nasal structures, it is found to lead down to the external and internal jugular or other secondary lymphatic chains on its way to the deep cervical chain of lymphatic glands.[2] Drainage from the teeth, tonsils, the nose and its sinuses finally comes down into the common descending chain of deep cervical lymphatics. The case reports telling of nodules which may be felt in the sternocleido mastoid, the trapezius and the levator anguli muscles strongly indicate the presence of enlarged glands in these regions. There is ample clinical evidence to support the view that enlarged lymphatics may give rise to pain. Hilton recognized pains in these regions as due to lymph gland involvement many years ago. The pain in the neck and shoulders may not be coincident with the onset of the nasal condition, but may come on days or weeks later. The pain in the region of the peripheral distribution of the cervical nerves may occur at a time when there is no pain whatever in the nose or eyes. It seems more reasonable to attribute the pain in regions remote from the nose to inflammatory or other pathological processes involving the nerves at those points in their course which lie close to the enlarged lymph glands than to some mysterious effect transmitted to the cervical nerves through the devious and even nonexistent connections of the

sympathetic system. I do not believe that the hypothesis of referred pain, as it is commonly understood, applies here. But I would pause to remark that taking out of the category of referred pain one group of cases in which only a most labored and highly improbable juggling enables us to arrive at any sort of an explanation, does not necessarily undermine the truth of the hypothesis in other cases of pain of obscure origin. I am merely endeavoring to find out whether the hypothesis of referred pain explains the findings in these cases of disease of the nasal tract. I have indicated that I do not believe that it does apply here. I am inclined to believe that it does hold in some other groups of cases.

If the view which I have taken of the mechanism of pain in the neck and the shoulder and the back of the head is correct, we must conclude that there is a very wide extension of disease processes located primarily in the nasal passages or sinuses. This need not surprise us in the least. We have long been harangued to have our teeth extracted because of the widespread, or supposedly widespread, effect of apical abscesses. We have been urged to have our tonsils removed for the same reason. But the benefits accruing from one or both of these procedures are often long delayed or disappointing. Neuritis of the fifth or of the eighth cranial nerve does not appear to be benefited by either procedure. Whence come the disease-producing agents responsible for these neuritides? For the auditory neuritis we do nothing. For the trigeminal neuritis we divide the sensory fibres in the maxillary division, but this is merely a palliative measure. Treatment of the underlying conditions in chronic cases is not done, for we do not know what these underlying conditions are. The ganglion of the trigeminal shows no changes. This is to be expected. The processes which are responsible for setting up the pain must be peripheral to the ganglion or else surgical procedures would be ineffective in stopping the pain. If the peripheral course of the trigeminal nerve is considered, we are led to suspect some primary condition in the nasal passages or its sinuses as the most probable cause of the trigeminal neuritis. It is possible, too, that some pharyngeal condition is responsible for the auditory neuritis. It is, furthermore, possible that some of these unknown and, at present, unrecognized conditions in the nasal tract or the nasopharynx may be responsible for the disappointments which sometimes attend tonsillar and dental operations. Will the amputation of the nose be demanded as a remedial measure?

I do not make this remark merely to be facetious, but to show the futility of some of our present methods of treatment. Resection of

the trigeminus does not, in all probability, stop the processes which set up the neuritis. In fact, the return of the pain in those cases in which there is regeneration of the trigeminus after resection would seem to show that the peripheral processes go merrily on. Nor will injection of the sphenopalatine ganglion with alcohol do more than stop the pain. It is true that these procedures can be carried out with little risk and that they give the patient relief. But the fact that the underlying conditions which set up the neuritides still persist offers a challenge to the therapeutist which cannot be overlooked. Some other form of therapy based on a more searching and deep-going study of disease conditions in the nose and its sinuses, not omiting the field of its lymphatic drainage, seems indicated. General pathologists have studied the axillary lymphatics in infections of the hand and arm, and the lymphatics of the thorax in metastases of cancer of the breast, but they have remained strangely aloof from a similar study of the retropharyngeal lymphatics in diseases of the throat and nose. The rhinologist and the laryngologist have had to make their own way as best they could. The recent successful efforts of the otologists to raise funds for research show that they appreciate the importance and the gravity of their problems for general medicine and surgery as well as for their own specialty. May their efforts lead to happier results.

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Columbia University.

FIVE MASTOIDECTOMIES AND A DECOMPRESSION OPERATION ON THE SAME PATIENT. PRESENTATION OF PATIENT.*

DR. PHILIP S. STOUT, Philadelphia.

From time to time patients return years after we have operated upon them and their history of the things that have occurred in the interim is often most instructive. We can see where we have failed, as well as where we have been successful and only a person who is deficient in intellect will close his eyes to the facts that present themselves. The followup system now practiced in some of our hospitals will do much to bring out these facts. We would suggest that whenever possible all cases discharged as cured (whether that particular term is used or not) be followed up. This would be the greatest help to bring out the actual results, good, bad or indifferent, and prevent the same errors being made over and over again.

Case: Bertha B., age 30 years, white, American, single; residence, Philadelphia; occupation, practical nurse.

Family History: Born in Philadelphia. Father died at the age of 67 years. Mother living and well, age 66 years. Two brothers living and well. Two sisters and one brother died in childhood with diphtheria. No chronic diseases in the family history.

Medical History: Had whooping cough in infancy. When she was 6 years old she was accidently thrown from the roof of a one-story back building of her home. She landed on some bricks, striking the back of her head. She was taken to the hospital, where she remained for several days, part of the time in an unconscious condition. Her mother was told that she had concussion of the brain, but no fracture. She got over this and the only ill effects were that she would throw her head from side to side while awake. This lasted for one year. She had no other illnesses until at the age of 9 years she put a coffee bean into her right ear canal. She was taken to the hospital to have it removed. After its removal the right ear discharged for eight months. She was under treatment in an ear dispensary during that time.

At the age of 10 years she accidently fell down in a grocery store and struck her right ear against a meat block. Blood flowed from

*Presented before the Philadelphia Laryngological Society, Feb. 7, 1928.
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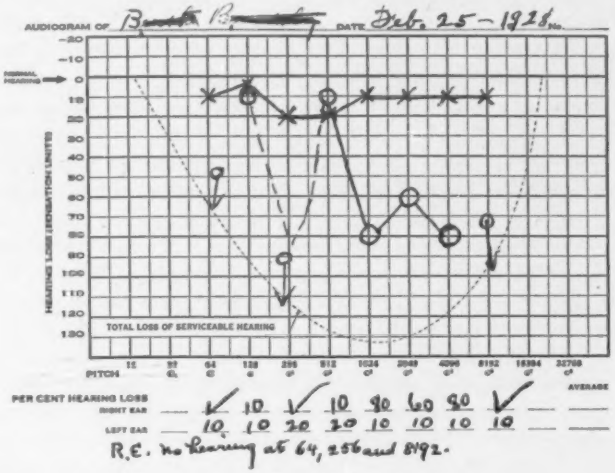
the ear. After this she began to have pain over the right mastoid area, some discharge from the right ear and some deafness was noted. She refused to go to the hospital for observation.

She was advised by the school physician to have her tonsils removed. She happened to come to the dispensary where the writer was at that time, 1907. Under local anesthesia with the tonsillotome a tonsillotomy was done. This was the operation used at that time. Needless to say that 10 years later, 1917, after an attack of diphtheria, she was compelled to have the tonsils properly removed. This was true of nine out of ten who were operated upon in those days with the tonsillotome, yet it was almost impossible to get the advo-



cates of tonsillotomy to see the error of their way. If there had been a followup system, overwhelming evidence might have aroused them from their mental lethargy.

About this time, 1917, she developed appendicitis and was operated upon. Nine months later she had a second abdominal operation for adhesions.

In 1920, the patient had pain on the right side of the head, seeming to concentrate around the right ear. She again came under the care of the writer and after study of the case with Dr. Keelor it was decided to do a modified radical mastoid on the right side. X-ray showed sclerotic bone, which was found at the time of operation. Drumhead perforation, very slight discharge, no foul odor

hearing fairly good. For a time after the operation great relief was experienced. Later her pains returned and she went to another hospital and they operated on the other, left, ear, simple mastoid. In 1922, pain again developed on the right side and she was sent by the nurse in the department store where she was then employed to still another hospital, where they again operated upon the right mastoid. Some time later, 1923, the pains on the right side of the head returned and she was again sent back to the same hospital and they again operated on the right mastoid. This made three operations on the right side and one on the left. See the confusion? These four mastoid operations for pain were done in three different hospitals by at least three different operators.

Some time later, 1923, while crossing the street she was knocked down by a touring car, taken to a nearby hospital, where it was found that she had a fractured skull; the fracture seemed to extend down to the operated field of the right mastoid. A decompression operation was done. She was quite ill and was in the hospital for about one month. She found that she had some weakness of the right leg and she dragged her right foot for about three months. That cleared up entirely.

In 1924, she had acute frontal sinusitis (?). She was taken to a hospital, where she was operated upon intranasally. Was in the hospital for two weeks. Several months after this she developed symptoms of most profound intracranial pressure; intense headache, nausea, vomiting. The writer was called in at this time to see her. Her condition was most alarming. She walked the floor with staggering gait, held her head because of the intense pain, had vomiting and nausea. She was sent to the hospital where the decompression operation had previously been done. They wanted to operate again immediately but the patient, for once, refused. She was so sick she said she would rather die. They used spinal puncture and all other means but operation to relieve the intracranial pressure. She finally got over it and left the hospital some weeks later in fairly good condition.

She went to the seashore and took a position as nurse in a children's home. She continued this work until she developed scarlet fever, April, 1927, and she was in a municipal hospital for some weeks. After this the right ear began again to give trouble. She developed one abscess after another. These would give her most excruciating pain, during which times she would show signs of intracranial involvement; headache, nausea, dizziness, at times becoming almost unconscious. Someone sent her back to the hospital where

they had twice operated on her right ear, but they were unwilling to operate again because at the last operation, on account of bleeding, probably from the sigmoid sinus, she had to be transfused several times to save her life.

November, 1927, she again came under the care of the writer. At this time she had just gotten over one of these abscess spells, more than a teaspoonful of pus had discharged from her ear when the abscess broke. She was sent to the Graduate Hospital (formerly the Polyclinic Hospital) for observation and study, and the X-ray showed a necrotic area in the upper portion of the mastoid area, posterior root of zygoma, where the fracture seemed to extend to the operated field. Hearing in the right ear was practically nil at that time. With the recurrence of these abscess formations and an area of necrosis in the X-ray picture, it was decided to operate again on the right ear. This made the fifth mastoid. The operation disclosed, as the X-ray had shown, an area of necrosis, which was carefully curetted away. Further, it was found that the sigmoid sinus was adherent to subcutaneous tissues and it was necessary to incise it to properly finish the operation. No difficulty was encountered to control hemorrhage, either at the time of operation or later. At the suggestion of Dr. Gleason a very wide plastic was done, the other plastics having given away completely. She made an uneventful recovery, she is free from pain at the present time, and it gives me great pleasure to present her to you this evening. In the light of subsequent events we doubt if we would have performed the first mastoid operation, which was done principally to relieve the pain she complained of. The relief she got from these operations was transient. Semicircular canals are active. Audiometer hearing chart submitted—made after the last operation.

Medical Arts Building.

INFECTIVE LATERAL SINUS PHLEBITIS. REPORT OF INTERESTING CASE, WITH RECOVERY.

DR. V. K. HART, Statesville, N. C.

This case is presented because of: *1.* Unusual clinical course. *2.* It is most interesting bacteriologically.

Long case reports are tiresome. Therefore the patient's history and findings are abstracted briefly. Girl, age 10 years. Seen first Oct. 28, 1927, with acute right mastoid—pain, edema, tenderness, temperature 102 (m). The drum was not bulging nor was there a history of aural discharge. Operated at once. Lateral sinus was partially uncovered because of overlying friable bone. Discharged Nov. 7, 1927.

Readmitted Nov. 10, 1927 (second week of convalescence), with temperature 102⅖ (m). Temperature varied from 102 to 105 (m). On Nov. 11, 14 and 16 there were spontaneous hemorrhages from sinus necessitating packing of wound. Sinus was packed off under light chloroform anesthesia Nov. 20, 1927. (Internal jugular was not ligated at this time because of negative blood culture.) Typical erysipelas developed about wound postoperatively. Recovered promptly after erysipelas antitoxin intramuscularly.

Septic temperature continued. Patient reoperated Nov. 27, 1927, despite persistently negative blood culture. Under local anesthesia, the internal jugular and facial veins were ligated and severed. The mastoid incision was carried back over the lateral sinus, sinus opened, external wall of sigmoid sinus partially resected. Sinus opened in both directions until free bleeding occurred from lateral sinus posteriorly and inferior petrosal below. Iodoform packs placed in sinus and between bone and dura above and below. (These were removed gradually during the next two weeks.) The neck wound promptly broke down and discharged but cleared up with Dakinization.

A very stormy convalescence followed of 46 days' duration. The temperature varied from 98 (m) to 106 (m). On Jan. 2, 1928, the temperature came permanently to the normal line. Steady improvement followed and the mastoid wound closed by granulation.

Therapeutics: Only a few essential things were emphasized, as in any sepsis: *1.* Fluids ad libitum with enough nourishment if possible to make about 3,000 calories for 24 hours. *2.* Ice caps. *3.* Digitalization. *4.* Blood transfusions. (Three of these were given:

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first, 150 c.c.; second, 150 c.c.; third, 275 c.c.) *5.* Quinin dihydrochlorid was given empirically in later stages in doses 7½ grains intramuscularly to reduce temperature. Sometimes as high as 30 grains were given in 24 hours.

Laboratory: These things are of interest: *1.* Persistent secondary anemia, hemoglobin ranging from 48 to 68 per cent. *2.* A tendency to low white count until secondary abscesses developed (four of them) at sites of quinin injections. Then followed a leucocytosis. *3.* Nine negative blood cultures; one positive for Hoffmann's bacillus. This latter was considered a contamination. All were taken at the peak of temperature. *4.* Nonhemolytic streptococcus was recovered on culture from *a.* mastoid wound and ear; *b.* neck; *c.* from secondary abscess on arm.

Comment: Why a persistently negative blood culture? The writer has had a case of lateral sinus infection where a positive blood culture for streptococcus was gotten every time it was taken. This was a case of frank thrombosis as demonstrated at operation.

It seems, then, that a clean cut distinction must be made between septicemia (organisms multiplying in the blood stream and therefore constantly present) and bacteremia where there is a periodic fluiding of the circulation with organisms, but where the body defenses are sufficient to conquer them until another invasion takes place. Either may give, of course, a positive blood culture, but the likelihood of such a positive culture is greater in septicemia.

If a bacteremia, where were organisms coming from after packing off sinus and ligating the jugular? They were there because they were present in abscess precipitated by quinin injection and consequent tissue devitalization. The answer is a retrograde flow of blood from lateral sinus, particularly if operation does not extend behind point where superior petrosal sinus joins lateral sinus. Furthermore, an endocarditis did not and has not developed.

The work of Ottenberg[2] in taking cultures from both internal jugular veins is strongly suggestive of a retrograde flow of blood. In two out of three cases reported the colonies were more numerous on the well side.

Fenton[1] reports a case of metastasis 20 days after ligation. The first abscess was opened in this case 30 days after ligation.

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Davis Hospital.

FIBROMA ARISING FROM THE JUGULAR BULB AND INVADING THE MIDDLE EAR AND EXTERNAL AUDITORY CANAL.*

DR. GEORGE W. MACKENZIE, Philadelphia.

An exact duplicate of the case to be reported was not found in the literature by the writer nor by a professional reference seeker, nor could a case of the kind be recalled by any of the Vienna School of Otology with whom the writer has been in touch.

There are so many odd and interesting phases concerning the case that it is quite impossible to cover them all in a title that would not be confusing. It is better, therefore, to limit the title to the fundamental condition in the ear and refer to the several other phases as they develop in the report of the case or in the comments.

These phases include: *1.* A dehiscence in the bony floor of the tympanic cavity, permitting an exposure of the jugular bulb surmounted by a thin (not more than 3 m.m. thick) fibroma; *2.* hemasiderotic staining of the mucous membrane lining the mastoid cells; *3.* severe hemorrhage on two occasions, once when a piece of the growth was removed from the depth of the external auditory canal for biopsy and again at the time of the mastoid operation performed for the removal of the tumor; *4.* unusual, but instructive Roentgenological findings. A discussion of any one of these phases would be sufficient in itself for a thesis.

The case, Miss A. M. F., age 18 years, first seen March 17, 1923, was referred by her father, a physician. Tonsils were removed at the age of 5 years; however, there is a stump remaining which the father thinks might be responsible for the present impairment of hearing in the right ear. She has been having attacks of hay fever for the past four summers. She is not susceptible to colds. There is impairment of hearing on the right side only. She has never had discharge from either ear. She complains of "ringing" in the right ear. There is also a slight discomfort at times in the jaws and lower part of the throat (indescribable in character). She has never had any dizziness.

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*Read before the Philadelphia Laryngological Society Meeting, Jan. 3, 1928.

Editor's Note: This ms. received in The Laryngoscope Office and accepted for publication Jan. 26, 1928.

Functional Hearing Test:

Right Ear.	Weber	Left ear not lateralized
	Schwabach	normal
short 3″ (?)	Rinné	+47″
+35″	Air	normal
short 5″	C_1	normal
slightly short	c_4	normal

The Schwabach taken on the left mastoid is referred to the right side.

Examination of the Nose: The mucous membrane on both sides is rather paler than normal; moderate deviation of the septum to the right side; the middle turbinate is barely visible on either side before shrinking the mucous membrane, because of the septal deviation to the right side and the inflammatory swelling on the left side. There is a spine along the suture line on the left side. There is no gross evidence of sinus disease on either side by rhinoscopic examination.

Examination of the Mouth and Fauces: Teeth are in good condition; tongue is normal; anterior faucial pillars redder than normal and slightly swollen on the free edges; small remnant of tonsil on the left side sealed over by adherent pillars. There is a slight degree of secondary catarrhal pharyngitis.

Otoscopic Examination: A. D. Tympanic membrane is intact, dull, somewhat opaque and waxy looking. It is impossible to discern the long process of the anvil through the membrane as in the case of the normal ear. The superior-posterior quadrant of the membrane appears to be slightly wrinkled, as though it were unduly flaccid. There is an increased vascularity of the membrane in this quadrant, due possibly to irritation from overmanipulation with the Siegel otoscope. With inflation after the Politzer method the membrane moves outward over a fair area and goes back to its primary position promptly. The hammer handle appears to be slightly foreshortened.

A. S. The tympanic membrane is intact, brilliant, no retraction, the long process is visible through the normally translucent membrane. The mobility with the Siegel instrument is normal; likewise, with inflation after the Politzer method.

The patient reported again on Sept. 7, 1923, suffering from an attack of hay fever. At this time a second functional hearing test was made, resulting in the following findings:

Right Ear.	Weber	Left Ear.
lengthened 10″	Schwabach	normal
—20″	Rinné	+40″
short 40″	Air	normal

May 7, 1924: The patient complains of nervousness. She has been using oil in the right ear to soften up what someone else had thought was hardened cerumen. After cleaning the canal of oil, a small, red swelling can be seen in the depth of the canal. It looks not unlike a polyp, however, rather less embossed. This swelling appears to be at the level of or very slightly external to the surface of the drumhead. A small crescentic-shaped area of the normal drumhead is to be seen just above the swelling. The patient was next referred to Dr. J. W. Post for Roentgenologic study of the mastoids. His report, under date of May 14, 1924, reads as follows:

"Roentgenologic examination of both mastoid processes of Miss A. M. F. reveals a normal pneumatic mastoid process and cellular structure on both sides."

A third functional hearing test was made on May 20, 1924, resulting in findings about the same as those of the last examination.

The patient reported once or twice a month up to July 14, 1924, when she claimed that her ear condition was much improved.*

Aug. 4, 1924: She reports having had an earache that was never experienced before. Upon questioning her she admitted that the earache was bilateral; besides, it was attributed by her dentist to the cutting of the third molars. The otoscopic findings recorded by Dr. W. G. Shemeley reads: There is a tumefaction along the floor of the right canal, purplish-red in color, extending almost to the upper periphery of the tympanic membrane. With a blunt silver probe it felt rather firm along the floor, but soft on the upper surface.

The patient reported again on Aug. 28, 1924; Sept. 3, 1924; Sept. 22, 1924, because of hay fever.

On Sept. 22 and again on Oct. 27, 1924, functional hearing tests were made. On both occasions the findings were much the same. That is, Weber referred to the right side. The hearing to air conduction moderately impaired to all tones on the right side and the Rinné negative. Normal hearing on the left side.

She reported off and on at odd intervals, sometimes telling of improvement and other times not. Some of the slight discomfort was rightly or wrongly attributed to the cutting of the third molars, which the dentist felt impelled to lance on more than one occasion.

*At the time of this report, the writer is unable to say why the patient should have reported her ear condition as much improved. It is possible that the tumor, prior to July 14, 1924, had been exerting a pressure on the walls of the tympanic cavity, and the drumhead which was intact up to this time yielded to the pressure of the growth that had previously been confined in the middle ear in a manner similar to that which occurs in the case of acute middle ear suppuration when the drumhead ruptures spontaneously.

On Jan. 26, 1925, the father of the patient removed a large scale (¼-inch square) from the external canal of the right side. On this occasion the extension of the growth externalward into the canal was quite evident. It was red in color and felt firm, but the firmness was not that of bone, as was suspected on one other occasion. There was a questionable pulsation to the growth which was not confirmed.

Feb. 6, 1925, the patient went through a septum operation with no discomfort and with good results so far as nasal respiration was concerned.

April 6, 1925, she reports that for the past ten days she has had intervals of pain in the right temporal and frontal regions. She also has a cough, which she claims starts from a "sensation" arising in the right ear.*

She continued to report at wide intervals up to April 27, 1926, when she was again referred to Dr. J. W. Post for Roentgenologic study of the mastoids. His report under the same date reads as follows:

"Roentgenologic study of both mastoids of Miss A. M. F. reveals evidence of the following: Left mastoid: Large-sized process of the small cell type and showing a well defined cellular structure of a pneumatic type. Right mastoid: Large-sized process of the small cell type and showing a generalized increase in the density throughout the entire cellular structure, but particularly so in the region of the mastoid antrum and between the posterior wall of the canal and the anterior wall of the sinus groove. The cell trabeculation can still be observed, and I do not see any of the characteristic evidences of active mastoid disease. The cortex of both processes is of tissue paper thinness. The appearance of this right mastoid rather impresses me of a low grade chronic process of a sclerosing type."

Nov. 2, 1926: Functional hearing tests reveal a slight increase in the deafness on the right side, of the mixed type. Normal hearing on the left side. At this time there was crossed conduction to the right side when taking the Schwabach on the left mastoid.

Nov. 30, 1926: Patient reports having had headache for the past two weeks, for which she was treated by Dr. Wm. Ivins, of Trenton. He used the mercury vapor quartz light in the treatment with apparent benefit. The patient is rather nervous and tends to be tearful. The otoscopic appearance of the right ear shows the same growth as on former occasions, but little changed except for slight increase in its size.

*Her explanation may be correct since reflex cough from irritation of the external auditory canal is not uncommon.

April 19, 1927: She reports that several times recently there was a slight bloody discharge from the right ear. The blood did not run out of the ear, but the ear felt warm to her and when she mopped it with her handkerchief there was a small spot of blood. At times there were four or five such spots. Otoscopic examination showed the growth questionably larger and slightly reddened on the upper surface, suggesting the location from which the blood transuded. To delicate palpation with the probe the growth at this time was of the consistency of a fibrous polyp.

May 12, 1927: The patient claims that the ear has been discharging a thin fluid. There is no pain to speak of in the ear. Otoscopic examination reveals the presence of a very slight amount of thin serous secretion. The tumor still resembles a polyp. Dr. Shemeley, with the consent of the writer, removed a piece of the growth for

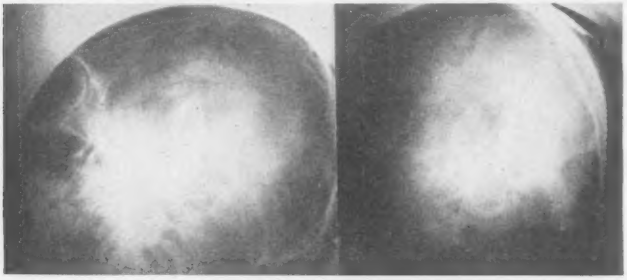

Fig. 1. May 14, 1924: Roentgenologic examination of both mastoid processes reveals a normal pneumatic process and cellular structure of both sides. (Legend by Dr. Post.)

biopsy. The writer not having been present at the time, will allow Dr. Shemeley to report his experience himself.

"The patient was prepared for the removal of a piece of the growth for biopsy by dropping a solution of 2 per cent butyn around the growth which presented itself in the external auditory canal of the right ear. A Krause nasal snare was used to obtain the specimen, through an otoscope with a large opening. The loop of the snare was passed over about 6 m.m. of the growth. The snare was then closed, at which time the patient jumped away from the restraining hands of the nurse, which resulted in a slight tearing action. Immediately, a stream of blood about the size of a goose quill spurted from the ear. The flow was continuous and with sufficient force behind it to cause a slight arching of the stream. Gauze was seized and immediately pressed into the lumen of the right external auditory canal.

Around this was pressed an additional packing of 1-inch iodoform gauze. A folded gauze pad was placed above these two packs and over the lobe of the right ear and pressure was applied by means of a tight bandage. This proved sufficient to control the bleeding. The packs were removed gradually; the last piece of gauze was taken out about one week following the removal of the specimen. Except for the discharge of a slight amount of bloody serum on one or two occasions there was no further bleeding from the right ear."

She was next sent to Dr. Post for further Roentgenologic study. His report reads as follows:

"May 16, 1927: Roentgenologic study of both mastoids of Miss A. M. F. reveals evidence of the following: Left mastoid: Large-sized process of the small cell type showing no evidence of pathology.

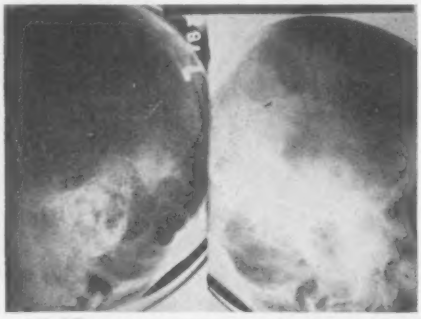

Fig. 2. April 27, 1926: Left Side: Normal small cell mastoid process. Right Side: Shows a generalized increase in density of the anterior portion of the anterior wall of the sinus groove and the posterior wall of the canal. The cell trabeculations can still be observed. There is no appearance of active mastoid disease at this time, although the general appearance would suggest a sclerosing type of pathology. (Legend by Dr. Post.)

Right mastoid: Large-sized process of the small cell type showing a generalized increase in density, especially over the anterior portion. There is no change in the cell trabeculations, nor can I see any evidence of bone resorption at any place. The appearance of this mastoid would give the impression of a pathological lesion, but it is very hard to definitely interpret the pathology when one considers the length of time over which it extends. It is rather hard to conceive of a pathological lesion in this right mastoid over this long period without evidence of bone changes."

May 17, 1927: The patient has been having pieces of gauze pack removed from the external canal of the right ear from day to day since the severe venous hemorrhage that followed the removal of a piece of the growth on May 12, 1927.

May 23, 1927: Functional hearing test reveals the presence of the Weber-Schwabach paradox, in that the Weber was referred to the right side in spite of the fact that the Schwabach on the left mastoid was 10 seconds longer than on the right mastoid. Furthermore, when the Schwabach was made on the left mastoid there was cross-conduction (lateralization) to the right side; suggesting the presence of something within the right mastoid spaces that interfered with the conduction of sound waves emanating from the surface of the right mastoid, reaching the perceiving apparatus on that side.

June 4, 1927: She claims that the throbbing sensation she had been having in the right ear has improved. She denies ever having had dizziness.

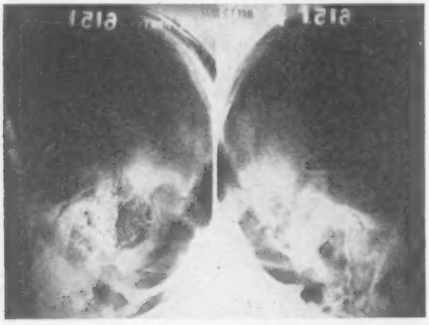

Fig. 3. May 16, 1927: Left: Normal mastoid process. Right: The generalized increase in density can still be observed over the anterior portion of the process with no change in the appearance of the cell trabeculations. There is no evidence of bony resorption at any place. (Legend by Dr. Post.)

Functional Hearing Test:

Right Ear.	Weber*	Left Ear.
lengthened 8″	Schwabach	lengthened 3″
—38″	Rinné	+28″
short 76″	Air	normal
very short	C_1	normal
slightly short	c_4	normal

The pronounced Weber-Schwabach paradox that was present at the last examination is absent at this visit.

Examination of the Vestibular Apparatus: When looking to the extreme right side there is a mixed rotary horizontal nystagmus to the right side of slight degree. When looking to the extreme left side there is a mixed rotary horizontal nystagmus to the left side of slight degree. The nystagmus to one side is no more intensive than

*Weber is referred to the right side when the fork is placed on the left mastoid.

to the other side. When looking straight ahead there is not the slightest evidence of nystagmus.

Turning Test: After 10 turns to the left, with the head erect, horizontal nystagmus to the right lasts for 24 seconds. After 10 turns to the right, with the head erect, horizontal nystagmus to the left lasts for 22 seconds.

June 8, 1927: The patient was operated radically for the purpose of removing the growth in the middle ear and external auditory canal. The operation was performed under ether narcosis administered by Dr. J. D. Rowland. Typical retroauricular incision, 4½ c.m. long. Separation of the soft parts, including the periosteum, from the underlying bone. The mastoid was opened with a No. 12

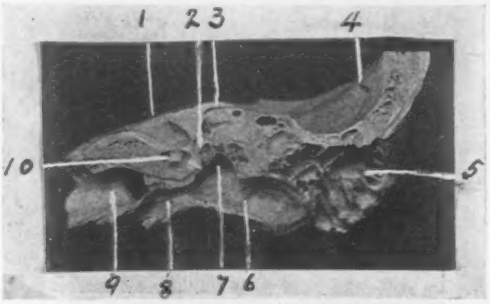

Fig. 4. Photographic reproduction of a horizontal section through the left temporal bone from one of the author's collection reveals a pronounced dehiscence in the inferior wall of the tympanic cavity. (1) Internal auditory canal. (2) Vestibule. (3) Vestibular acqueduct. (4) Internal opening of emissary vein. (5) Mastoid cells. (6) Bony external auditory canal. (7) Dehiscence in the posterior part of the floor. (9) Horizontal portion of carotid canal. (10) Cochlea.

Alexander chisel when no pus was found; instead there was a slight amount of yellowish-stained, transparent fluid issuing from the deep cells under pressure and pulsating. Instead of following the usual method of entering the antrum first, the cells were opened superficially throughout the whole mastoid, leaving the antrum until quite late. The most striking thing about the mastoid cells was the peculiar greenish-yellow discoloration of the mucous membrane lining them that resembles the stains from blood salts, particularly iron salts (hemasiderosis). The deeper the cells were opened the more convinced the writer felt that the discoloration was due to the blood salts. In some places the discoloration took on a dark greenish color. Two other striking features found were the absence of pus and the normal condition of the bone. The posterior wall of the canal was not

removed until very late in the operation for fear of provoking bleeding. The radical operation was completed after the Kuester-Bergman method, when a cyst-like growth with smooth, but unpolished surface presented itself. It was elliptical in shape, long axis running lengthwise with the canal. It filled the osseous canal completely. In its more external portion was an extra lobule, polypoid in appearance, which seemed to be entirely distinct and different from the larger, more smooth mass. The tumor mass was carefully separated from the surrounding walls of the canal, excepting at the lower margin of the annulus tympanicus, in order to feel certain that the major part of the mass was exposed. It was then possible to see the dull red-colored tumor with its rough surface resting upon the blue, smooth surface of the deeper lying jugular bulb. The tumor mass was engaged in the snare and removed, when there followed a profuse

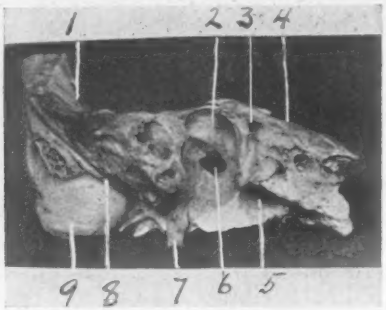

Fig. 5. Photographic reproduction of the same specimen as in Fig. 1, viewed from below. (1) Posterior skull fossa. (2) Jugular bulb. (3) Opening of the cochlear acqueduct. (4) Internal auditory canal viewed edgewise from below. (5) Carotid canal. 6) Dehiscence in the jugular bulb. (7) Styloid process. (8) Digastric groove. (9) Mastoid tip.

flow of dark venous blood. The stream was about the diameter of a lead pencil. There was no pulsation to the stream. Everyone present felt satisfied that the bleeding was from the jugular bulb. It required considerable packing, both through the canal and posteriorly to control the bleeding. The patient lost more than a cup of blood within a few seconds before the bleeding was entirely under control. A plastic operation after Panse had been made prior to the removal of the mass. During the operation the facial canal was opened, but the nerve was not severed, as proved later by the absence of facial nerve involvement.

Two days later the writer left for Europe, when Dr. Shemeley took charge of the after-treatment. On account of the risk of sec-

ondary hemorrhage from the jugular bulb he did not remove the dressing next to the opening in the jugular bulb until June 27. From that time on the patient made an uneventful recovery.

Functional hearing tests, Aug. 29, 1927, revealed the hearing loss and character of the loss about the same as before the operation.

Dec. 28, 1927: Functional hearing tests show some improvement in hearing. In fact, it is much better than before the operation. The retroauricular wound has long since healed over. There is no aural discharge. The healing has been ideal from every angle.

The biopsy report of Dr. George Hopp follows: "Microscopic examination of sections shows a connective tissue tumor with intact epithelial covering. The growth is almost entirely cellular, the cells

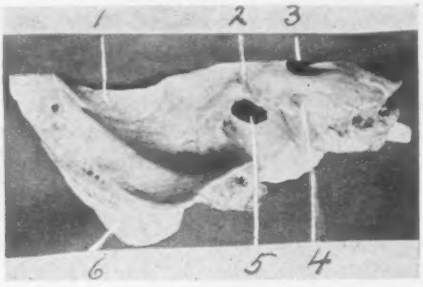

Fig. 6. Photographic reproduction of the same specimen as in Figs. 4 and 5, viewed from the posterior aspect. (1) Sinus groove. (2) Depression for the endolymphatic sac. (3) Internal auditory canal. (4) Opening for the cochlear acqueduct. (5) Dehiscence in the jugular bulb. (6) Mastoid process.

are fairly uniform in size and show no mitotic figures. Vessels are numerous and dilated. Specimen may be a sarcoma, but the general appearance is that of a benign growth."

COMMENTS.

1. Dehisence of the floor of the tympanic cavity or, more strictly, of the fossa jugularis must have been present in this case for the following reasons: *a.* The location anatomically of the major part of the swelling; *b.* the fact that such dehiscences do occur in this locality even though rarely, as shown by the specimen herewith presented; *c.* the smoothness of the surface of the sinus as compared with the relatively rough surface of the tumor that rested on the sinus; *d.* the profuseness of the hemorrhage, the dark color of the blood, distinctly venous and nonpulsating.

The jugular bulb presenting in the tympanic cavity through a dehiscence in the jugular fossa may be recognized otoscopically

through a normally translucent drumhead, by its bluish color, convexity upward and its location—posterior-inferior part of the mesotympanum. They may be obscured in the case of a drumhead made opaque by the presence of an organized exudate on its median surface or inflammatory reaction (myringitis), accompanying an acute middle ear suppuration. In the latter instance a deep incision of the membrane is likely to be followed by severe and obstinate bleeding.

2. Hemasiderotic staining of the mucous membrane lining of the mastoid spaces. The staining of the tissues of the body, especially the eye, is not unusual. The characteristic iron rust stain of the eye, especially of the cornea, is easy to recognize by oblique illumination. It can be recognized by its typical reaction in sections with a 2 per cent aqueous solution of ferrocyanid of potassium, known as the Berlin Blue method. The tissues may be stained not only by iron salts from foreign bodies, but also by the normal iron salts of the blood following hemorrhage into the tissues. The writer was privileged to see two cases of marked hemasiderosis of the drumhead. In one case he had the opportunity of seeing the case early and again years later. In the other case, seen with Dr. Zacks, a fellow member of this Society, the changes were late and fixed.

The writer never before saw a case of hemasiderosis of the mucous membrane lining the mastoid cells; nor is he able to locate another case in the literature. The color was not that of an old hemorrhage, like that of the other two cases, in which the drumhead presented a greenish-black color. The hemorrhage responsible for the mastoid cell staining probably occurred at the time Dr. Shemeley removed the specimen for biopsy. This is further supported by the fact that just after that time the patient presented the Weber-Schwabach paradox.

The writer begs to reserve the privilege of a fuller discussion of this phase of the case for some future time, when he hopes to present the subject of hemasiderosis more fully in connection with the reporting of cases of hemasiderosis of the drum membrane.

3. Hemorrhages, spontaneous and otherwise, from the ear is a subject in itself and deserves separate attention elsewhere. In this case the history tells of spontaneous bleeding, of small amount, not more than enough to spot a handkerchief, occurring late in the case, due apparently to an abrasion, possibly from the handkerchief used in mopping, for the abraded surface was seen otoscopically and abrasions do not occur except upon the application of some object capable of producing them. One thing is certain, and that is, the spontaneous bleeding did not come from the sinus, but from the super-

imposed fibroid tumor. Had it come from the sinus it would have resembled, in character and quantity, the gushing dark-colored blood observed when Dr. Shemeley removed a piece of the tumor and observed again at the time of the mastoid operation. The hemorrhage provoked on these two occasions was unquestionably jugular bulb bleeding.

4. Concerning the correlation of the preoperative Roentgenologic findings and the operative, the writer has asked Dr. Post to make his own comments and show the films.

"The correlation of my preoperative X-ray findings and the operative findings of the essayist upon this case are deserving of some comment and serve to emphasize several points in the study of mastoid disease.

"The first point which I should like to stress is the fact that the slightest change in the structures in and about the mastoid process can be depicted and interpreted on the Roentgenograms.

"Secondly, it is of infinite importance that the Roentgenologist be acquainted with the full clinical history without necessarily being informed as to the otoscopic findings. Lastly, and above all, it emphasizes the necessity for an intimate co-operation between the Otologist and Roentgenologist.

"For the analysis of the Roentgenological reasoning and interpretation in this individual case, I would refer you to the X-ray report incorporated in Dr. Mackenzie's paper."

5. It is interesting to note the occurrence of the Weber-Schwabach paradox just after the first severe hemorrhage which tells us that at the time of the severe bleeding from the canal, followed by the tight pack introduced into the canal to stop the bleeding, that some of the blood found its way into the mastoid spaces, which was responsible for the unusual Roentgenologic findings and the Weber-Schwabach paradox. Further confirmation of the recentness of the hemorrhage is indicated by the light (rusty) color of the hemasiderotic stain of the mucous membrane lining of the mastoid cells, while in old cases the staining is of a dark greenish color.

In reportng the case many unessential details have been omitted, besides the comments have not been as full as the circumstances in the case warrant. The writer hopes to fill out some of the details lacking in this report on another occasion in the presentation of one or more phases of this case as separate subjects.

1724 Spruce Street.

RELATION OF THE SINUSES TO THE GENERAL ECONOMY.*

DR. HARRY A. SCHATZ, Philadelphia.

The paranasal sinuses are lined by a very delicate, transparent membrane about 1-50 inch in thickness, which the naked eye can barely if at all differentiate from the subjacent periosteum.

Sinusitis is a pathologic condition depending on infection, but engrafted upon an underlying general physiologic disturbance, or perversion in metabolism. The latter is manifested by a hypersecretion of mucus by the mucous membranes wherever they occur, not all of them necessarily participating at the same time. This condition begins in early life—in infancy, perhaps; and lasts, shall we say, a lifetime? In common parlance, it is "catarrh". Its course throughout life is not uniform, exhibiting ups and downs, affecting now one and again another of the mucous membranes of the body. Various environmental factors, such as climate and seasonal changes, degree of sunshine, physical and mental fatigue, diet, exercise, infections, all play their part.

It is with special intent that infections are mentioned last. Except for the catarrhal infections which are usually epidemic in character, the ordinary infections concerned in suppurative sinus trouble play but a secondary, though not a minor, role. Without stagnation entailed by swelling and hypersecretion of mucus at the site of the sinus ostia there can be no sinusitis. Lothrop, in writing of his frontal sinus operation, states that in breaking through the interfrontal septum to enlarge the outlet from the diseased side he did not encounter any consequent infection in the healthy side. Comparison of the diseased lining membrane found when doing a radical sinus operation—whether frontal or maxillary—makes one wonder how such a profound change can occur in what is normally an insignificant tissue. There is a ballooning of the lining and the cavity is filled with a glairy mucinlike exudate with more or less pus, depending on the progress and type of infection. Interference with the venous outflow caused by pressure at the ostium is the only explanation of that, coupled of course with infection. Relief of this pressure is therefore the key to this situation. Surgery must be employed

*Read before the Philadelphia Laryngological Society, Nov. 1, 1927.
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in these cases, whether acute or chronic, where local and general remedies fail.

It is especially in the treatment of long-lasting chronic cases and those acute cases that are on the borderline of surgery that a consideration of the general or metabolic underlying factors are helpful.

The fluid media of our bodies are constantly enriched (or vitiated, as the case may be) by chemical substances, exogenous or endogenous or bacterial, that either serve as nutriment or detriment to the tissues, depending on whether they are intended as food or intended for excretion. Not only does the animal body eliminate through the usual or conventional eliminative organs, but at all times there is a state of osmotic tendency towards the excretion of poisonous or unwanted substances through every tissue in the body. Whether it will be through the skin, nose, throat, eye joint or heart, and so on, depend on various, perhaps unknown, factors.

One of these factors of prime importance must be the state of vascularity of the particular organ. Cadaver operations have taught me that the internal jugular vein is a very variable blood channel as far as size is concerned, but the carotids are more uniform. Imperfect venous drainage will entail stasis. If one inherits or develops a florid skin certain skin lesions are more prone to develop. Local irritative factors are of course also among the prime ones.

Psychic or emotional states are of importance. Just as we understand blushing of the face as an emotional manifestation, so we can conceive blushing in any organ or tissue. The intake of warm or spicy food causes a filling up of the nasal mucosa and a hypersecretion, if not under normal, at least in abnormal states of the nasal passages.

Another determinating factor is any gross anatomical deviation from normal, also local irritants of any sort. A few years ago Dr. Stauffer read a paper stating that an attack of acute coryza can occur regularly after eating a meal too rich in sugar particularly. Another writer wrote some years ago that the liver was at fault in sinusitis, and he recommended calomel and attention to the liver. In fact, it may be that the mucosa lining the gall bladder and cystic or hepatic ducts is affected by the same catarrhal excitant as the nasal. However, the nasal, pharyngeal, bronchial or tracheal catarrh is not so detrimental to the nutrition as that affecting the liver. Hence attention to the liver by purgation and a bland diet is very important.

Demonstration that the nasal mucus contains toxic substances, which are being eliminated through the mucosa, would be convincing,

but biochemistry at present is unable to do so with those subtle and elusive toxins of most interest to us. In fact, they have not yet been identified. But administer a dose of sodium iodid and test for it in the nasal mucus. It appeared about three hours after intake and continued to show even 19 hours after. The mucosa lining the middle ear should be the next one to test out. Its bearing on catarrhal otitis media is problematical, but interesting just the same.

The important first step in treatment is to assist the liver in its function as a destroyer of poisons or toxins. Flushing out the gastrointestinal tract and a bland diet, such as milk, crackers, fruit juices and, if possible, rest, sunlight or ultraviolet raying, have promptly relieved a boring pain in the head and orbit due to swelling and pressure in the ethmoidal sinuses and also caused a disappearance of pus. Sodium chlorid is also under suspicion, whether from its influence on osmosis or as a direct irritant because of its elimination through the nasal mucosa is to be investigated later. Also the diet must be a scanty one. In one case after 10 days of comfort, one meal with meat caused return of pain and pus. Several times at about weekly intervals meat even in moderation proved an exciting factor of the symptoms. The local evidences of swelling and inflammation also gradually subsided so as to make surgery no longer necessary. Gradual return of the digestive powers were met by increasing the amount and variety of foods, but meat in this case was for a long time taboo. How long is still indeterminate.

This attitude as regards sinus disease and nasopharyngeal catarrh seems to explain why we may see a patient, who has been cured of a suppuration in one sinus, return with trouble in another; or when a perfect submucous resection is performed and the patient is worse, or not benefited at all.

The participation of the ear as evidenced by tinnitus and impairment of hearing in this general catarrhal affliction has not been mentioned, though not forgotten, because not included in our title.

1737 Chestnut Street.

SYSTEMIC MANIFESTATIONS OF VINCENT'S INFECTION.*

DR. CHARLES FREDERICK TENNEY, New York City.

Vincent's infection is an acute infectious disease of the mouth and throat, due to the combined action of a spirochete and a fusiform bacillus with other associated organisms. Locally it is classified by inflammation and the formation of ulcerative lesions, large or small in extent, which may be located on the mucous membrane of the cheeks, gums, tongue or tonsils. Necrosis of the mucous membranes then takes place; this is followed by a grayish membrane at the site of the lesions. Constitutional disturbances follow this infection of greater or lesser severity, with enlargement of the lymph nodes. When this takes place, it is more apt to be associated with other mouth organisms, such as streptococci and staphlococci. The mild forms are more common and slower in their progress. They do not penetrate so deeply in the mucous membrane. The severe type has a more rapid onset and more pronounced constitutional symptoms.

This infection as described by Vincent probably received the term angina when the more active involvement of the lymphoid tissue was in the floor of the mouth and throat, causing a choking sensation, and making it more of the anginal type.

The conditions of Vincent's are similar and perhaps the same as trench mouth, which was so prevalent in the army. This has been described by Reasoner and Gill, of the medical corps. They quote Woods as stating "that in the mild forms the fusiform bacilli predominate, while in the more severe forms the spirochete seems to be in predominance."

During the past year in the laboratory of the Fifth Avenue Hospital, we have been carrying on some research work relative to Vincent's angina under the direction of Miss Pratt and Dr. Lintz. Fusiform bacilli were found in the throats of man in 100 per cent of the cultures examined; 100 per cent in the monkeys; 66 per cent in the guinea pigs, and 48 per cent in the rabbits. Two hundred human throats were cultured, about 50 of which were clinically normal individuals. The other 150 complained of colds or sore throat, or Vincent's was found as an accompanying sickness, associated with a primary disease. The isolated cultures were divided into two groups on the bases of sucrose fermentation and morphol-

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ogy. These two groups had no relation to the clinical condition. According to Miss Pratt, the complement fixation failed to distinguish any relationship between the strains, each antigen fixing complement with its homologous serum only, indicating that the bacilli formed a heterogeneous group. Animal inoculations showed that fusiform bacilli have a slight variable pathogenicity which is quickly lost. A few isolated strains, when inoculated into the buccal mucous membrane of a rabbit, produced a small punched-out ulcer, resembling that found in Vincent's angina. The predominant organisms then in Vincent's angina are fusiform bacilli and spirochetes. These also are associated with other mouth organisms, such as streptococci and staphlococci, and occasionally diphtheria bacilli.

During the past three years there have been twenty cases of Vincent's angina in the Fifth Avenue Hospital, all of which showed varying degrees of constitutional disturbances. Males and females were about equally affected. The ages of the individuals varied from 2 to 60 years. The symptoms were those of general malaise and temperature ranging from 98.8° to 106.6°. The average temperature of these cases ranged about 102°, and this lasted only from one to two days. The leucocyte count ranged from 6,000 to 90,000. (This last statement of 90,000 had better be explained as this was a Vincent's associated with a case of lymphatic leucemia.) The average leucocyte count was from 6,000 to 28,000, with a polymorphonuclear from 57 to 88. Smears were positive in 11, negative in four, and not done in five. Cultures were done in only three of these cases and were positive in three. Three of the cases died; one with a diagnosis of streptococcus septicemia, another with lymphatic leucemia and another of pulmonary infarct. The patients on admission had a temperature, which after treatment was started very quickly subsided, except in the fatal cases. They were quite comfortable, aside from the irritation of a sore mouth and throat. There was great discomfort in masticating the food when the lesions were around the teeth and considerable difficulty in swallowing when the lesions are in the throat or on the tonsils. When the involvement was on the mucous surfaces, more particularly around the teeth, the constitutional manifestations were more severe. Some weakness and exhaustion naturally followed this, and on some occasions, where the ulcers extended below the mucous surface, abscesses developed which had to be incised and took longer to heal. There is a characteristic odor to a Vincent's infection of the mouth and throat, fetid and musty in character. In the severe types of cases the infection lasts from five to ten days.

Undoubtedly the sodium perborate treatment of Vincent's is the treatment par excellence today. Dr. Bloodgood in a recent article in the *Journal of the American Medical Association* recommended that a thick paste of the chemically pure salt should be made with water and spread over these areas. The patient should hold the paste in the mouth for about five minutes. During this time it foams as a result of oxidation. Then the mouth should be rinsed, and the throat gargled with warm water. A thinner solution should be used as a gargle three or four times a day.

We are all familiar with the symptoms of follicular tonsillitis and know that the streptococcus is the predominating type of organism in this disease. However, many times fusiform bacilli and spirochetes are found in the cultures of these throats. Occasionally in a diphtheria throat, the membrane which closely resembles the growths of Vincent's, is many times confused with a Vincent's. The fusiform and spirochetes seem to be much less profuse in their growth when associated with the diphtheria bacilli than when associated with the streptococci, whose presence in increased numbers seems to make a more favorable field for the development of the spirochetes and fusiform bacilli.

Spirochetes and fusiform bacilli always seem to be present in the cultures taken from the mouths and throats of normal individuals. They probably form part of the mouth organisms that are always present. They are, however, apt to become more active in the presence of pyorrhea, decayed teeth or tonsils that are chronically infected. When these growths become manifest a diagnosis of fusiform bacilli and spirochetes can be made by direct smear, but should always be cultured, so that if diphtheria bacilli are present the disease may be recognized as early as possible. We have all had the experience of looking at these membranes and wiping them off to see if they bleed beneath the surface to make a clinical differential diagnosis between Vincent's angina and diphtheria, as the diphtheria membrane is much more adherent than the Vincent's.

In concluding I should say that the fusiform bacilli and spirochetes are present in all mouths and throats. They are more apt to become active in the presence of pyorrhea, decayed teeth, infected tonsils, and where streptococci predominate. They are not apt to become active in the presence of diphtheria bacilli. Vincent's angina is a disease, the membrane of which closely resembles diphtheria. The symptoms are sore mouth or throat, difficulty in mastication and deglutition, elevation in temperature and increase in the leucocyte count. The sodium perborate treatment should be tried in all cases.

Fifth Avenue Hospital.

VINCENT'S INFECTION OF THE MOUTH.*

DR. RAYMONDE ADAIR ALBRAY, Newark, N. J.

During the past eight or ten years there has been considerable discussion of the condition variously known as Vincent's angina, Vincent's disease, trench mouth, ulceromembranous stomatitis and a number of other designations. Prior to the World War it was rather infrequently encountered, although well established as a pathological entity, the disease and its causative organisms having been described by Plaut and Vincent in 1894 and 1896.

During the World War it appeared among the men in the armies in Europe and spread with amazing rapidity, incapacitating large numbers of the soldiers, and the name "trench mouth" was given it. In the army hospitals, conditions were such that the dentists and physicians could not institute proper treatment over a period of time sufficient to insure the eradication of the organisms of the disease from the mouths of the afflicted men. It was also impossible for the patients to give the amount of care and attention to the hygiene of the mouth, which is so very necessary to a successful outcome in the treatment. When the acute manifestations had been gotten under control, a great majority of the cases did not receive any further treatment, with the result that many of our soldiers upon their return to this country were carriers of the disease. Soon infection was communicated to members of their families and to others, until, in some sections of the country the disease had assumed almost epidemic proportions.

If Vincent's disease was confined to the mouth alone there would be sufficient reason for attaching considerable importance to it. The damage to the gums and to the alveolar process which accompanies and follows this infection is considerable. Teeth are frequently lost, and the destruction of the gum tissue and alveolar process around and between the teeth establishes food pockets, which sooner or later result in the further loss of teeth. In addition to this, it is not at all unusual for the organisms to spread from the mouth to other parts of the body, and by their growth and proliferation produce the typical gangrenous process for which they are noted, thereby causing irreparable damage and sometimes death.

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As you well know, the bacillus fusiformis and the spirochetes of Vincent's are recognized as the causative agents of the disease under discussion. In the *Jour. A. M. A.* for Sept, 16, 1922, Davis and Pilot present a study of these organisms, and I will quote from that paper several conclusions arrived at by these investigators.

"The occurrence of the two organisms in putrid pneumonia and lung gangrene has been noted. Rona especially has made important observations in this connection. We have observed a number of cases all with foul, fetid sputum teeming with B. Fusiformis and Spirochetes forms, together with streptococci.

"Infection with fusiform bacilli and spirochetes may be superimposed on pulmonary tuberculosis. This is illustrated by a patient now under observation, who is in an advanced state of tuberculosis and is expectorating very foul sputum. Cocci, together with B. Fusiformis and spirochetes occurred in great abundance in the sputum, which also harbored many clumps of tubercle bacilli.

"It should also be pointed out that syphilis is not in any way a factor in the above-mentioned infections."

Weaver and Tunnicliff, in 1907, in the *Jour. of Infectious Diseases* show "as the direct etiological factors in noma, B. fusiformis and spirochetes, some debilitating contagious or infectious disease, especially measles being a predisposing factor."

To quote again from the above-mentioned paper of Davis and Pilot, "Putrid otitis media deserves consideration here, since B. fusiformis and spirilla forms have been found by a number of observers in the discharges."

In the *Jour. of Infectious Diseases* for August, 1923, Pilot and Pearlman say: "Chronic fetid discharges from the middle ear contained fusiform bacilli, spirochetes together with streptococci, pneumococci and diphtheroids. Nonfetid discharges did not harbor the bacilli and spirochetes. The B. fusiformis and spirochetes appeared identical with those about normal teeth, tonsils, adenoids and in the putrid infections of the mouth and lungs."

In the *Jour. A. M. A.*, Sept. 16, 1922, Davis and Pilot state that "the introduction of exudate from Vincent's angina under the skin of rabbits will result in a few days in a putrid process. The presence of the associated cocci, especially streptococci, is no doubt an important factor in determining the character of the lesion and exudate. The cocci appear to be the more aggressive, advancing into adjacent tissue or organs, whereas the fusiform bacilli and spirochetes follow and cause the necrotic and gangrenous process." These investigators further state: "A large number of the various putrid

and gangrenous processes in which B. fusiformis and spirochetes play a role begin about the mouth and pharynx. Less frequently, they occur in the bronchi, lungs, pleura, middle ear, etc., parts further removed but still all more or less accessible to the infected material from the foci in the mouth and throat.

"In this type of infection, we find that persons suffering with a variety of diseases, poorly nourished and with unclean mouths are most often attacked. There appear to be some exceptions, however, to the foregoing statement, for outbreaks and epidemics of Vincent's disease are well known in which large numbers of normal persons without a recognizable predisposition are infected. To explain this, we may assume that, like other bacteria, B. fusiformis and spirochetes may take on a heightened virulence or at least take on a property which permits them to gain a foothold more readily in the tissues."

Klein and Blankenhorn in the *Jour. A. M. A.* for Sept. 1, 1923, state that "evidence is accumulating that pulmonary gangrene may be caused by spirochetes and fusiform bacilli aspirated from an unclean mouth". They cite four cases in which large numbers of the organisms were found in the sputum and which they believe were directly traceable to the spirochetes and fusiform bacilli that were aspirated from gangrenous ulceration in the mouth.

I have quoted at some length from these investigators to impress upon you the fact that Vincent's disease in the mouth is a condition deserving of serious and conscientious attention by every physician and dentist who is called upon to treat it. I also wish to emphasize that the insidious nature of this disease practically demands that smears be made from all soft tissue lesions of the oral cavity to determine the presence or absence of the organism of Vincent. Too often it goes unrecognized, or the seriousness of the condition is not appreciated, with the result that the patient is caused much unnecessary suffering, and not infrequently the functions of organs and tissues are seriously interfered with or are damaged beyond repair. At this point it may not be amiss to suggest the consideration of a possible diphtheria infection, especially when the lesions are on the fauces, tonsils or pharynx. It is especially important to take this into account in young patients. Mucus patches and other syphilitic manifestations in the mouth must also be thought of in connection with the diagnosis and treatment of Vincent's disease. It is well to have a Wassermann test made, at least in patients where the disease in running a severe acute course, for we have found a number of instances in which syphilis was undoubtedly a factor in the severity

of the destruction taking place. Another very important factor to be considered is that the organisms of Vincent may be found in the gangrenous mouth lesions which are sometimes associated with leucemia and pernicious anemia. I have seen several cases of each of these diseases in which smears from the lesions showed practically no organisms but cocci and those of Plaut-Vincent. This demonstrates the fact that a differential blood count is of great importance when there are large areas of gangrene in the mouth, even though smears seem to indicate that the trouble is Vincent's disease.

Case 1: D. J., male, age 40 years, presented with what appeared to be a mild Vincent's infection of the gingiva. Smears were taken and large numbers of Vincent's organisms found. The mouth was cleansed and a 5 per cent solution of salvarsan in glycerin applied. Improvement was marked and after two weeks the patient discontinued treatment. Several weeks later he again presented, this time with an ulcer about ¼-inch in diameter on the gum just below the lower right first bicuspid. Salvarsan was applied, but in 48 hours the lesion had spread so that the bone on the entire right side of the mandible and maxilla was exposed, and the entire inside of the cheek was a gangrenous ulcer. Smears were made and showed B. fusiformis and spirochetes of Vincent and cocci in tremendous numbers. A Wassermann was negative, but a differential blood count disclosed a severe myelogenous leucemia. By careful treatment we succeeded in checking the progress of the gangrene in the mouth and resolution was nearly complete in about three weeks, when the patient developed a colitis and died several days later. I have seen several other cases similar to the one just cited, with symptoms and findings about the same.

Stitt, in his "Practical Bacteriology, Blood Work, Parasitology", 8th edition, 1927, page 587, says: "An interesting feature of infections with these associated organisms is that they appear to provoke a lymphocytosis, the blood picture often being such that it is easily mistaken for that of acute lymphatic leucemia." I have seen no other reference to this observation by Stitt and it will be interesting to be on the alert to confirm it.

Case 2: R. L., female, age 30 years, presented for treatment of severe inflammation and ulceration of the mucous membrane of the gums and cheeks. Smears showed large numbers of Vincent's organisms and masses of cocci. The patient's color and general appearance suggested pernicious anemia and a blood examination confirmed the suspicion.

William Hunter, in the *British Medical Jour.*, of March 18, 1922, writing on the subject of "Pernicious Anemia and Septic Anemia", refers to the swollen and ulcerated tongue and gums which are symptoms of the disease, and stresses the importance of oral sepsis as a complication. He says nothing of the bacteriological findings in the mouth but does state that, "marked improvement and some recoveries have resulted from removal of the oral sepsis and treatment with salvarsan, neo-salvarsan and other arsenic compounds." This would seem to indicate the probable presence of Vincent's organisms in these mouths.

I have seen several other patients with pernicious anemia who had mouth symptoms simulating Vincent's disease, and in which the organisms of that disease were present, always associated with masses of cocci, which on culture, were found to be principally strep. viridans and staph. aureus.

The cases cited have impressed me with the importance of inquiring carefully into the general physical condition of patients with severe manifestations of Vincent's disease, as well as in those cases of the disease which did not respond readily to treatment.

Case 3: One of the most distressing cases of Vincent's disease I have ever seen was that of a young lady, age 20 years. She was partially paralyzed as the result of a railroad accident. The family was very poor and the girl under-nourished. The gums about her upper incisors became badly inflamed and very painful. A dentist was called to the house and at his advice the affected areas were painted with tincture of iodin, and a proprietary mouth wash was used. The condition spread and two weeks after the onset I was asked to see the case. There was no doubt as to the character of the disease and smears showed Vincent's organisms and cocci in tremendous numbers. Destruction of soft tissue had progressed to such a degree that much of the alveolar process was entirely denuded. A blood transfusion, and salvarsan both intravenously and locally, failed to check the progress of the gangrene and necrosis and the entire maxilla was exfoliated as a sequestrum. The alveolar process of the mandible was destroyed and the teeth dropped out. The infection got into the patient's eyes and they became gangrenous. Septicemia developed and the patient died several days later. Syphilis was not a factor in this case, as the Wassermann was negative, and while the patient was very anemic there was no evidence of leucemia or pernicious anemia.

Reports of two other cases may be included at this point to illustrate the value and necessity of early recognition and prompt treatment.

Case 4: Male, age 28 years, with severe diabetes. I had seen him several weeks prior to the Vincent infection, at which time his mouth was in excellent condition. He presented an acute Vincent's ulceration about a lower first molar, and stated that he had first noted the soreness two days before. The ulceration had exposed the alveolar process all around the tooth, which was so loosened that I removed it with my fingers. Naturally a patient with diabetes is a very poor subject when infected with a disease of this type. I consulted with his physician and we decided that small doses of salvarsan intravenously were indicated in conjunction with local treatment. This program was followed and the results were most gratifying, for we got complete control of the infection and destruction, and after two weeks of treatment could not find any B. fusiformis or spirochetes in his mouth, and there was no further involvement.

Case 5: Female, age 18 years, convalescing from a prolonged illness. Severe ulceration of the lips and gums developed several days after she had been kissed by a friend, who, by the way, later came to me for treatment for Vincent's. The physician of the young lady asked me to see the case with him. Four teeth were practically exfoliated and had to be removed, the ulceration had spread to the face and to the tonsils and pharynx. Salvarsan was used intravenously and locally and the patient made a good recovery.

From my experience I would say that when large areas have become necrotic as a result of the activity of B. fusiformis and spirochetes, aided by streptococci and other organisms, it is only a short step to the breaking down of the normal and natural defensive forces of the body, with resultant septicemia.

I have four patients who have Vincent's disease in a chronic form. One young man has never had a filling or a cavity in his teeth, and all of these patients have their full complement of 32 teeth in normal occlusion. They presented for treatment of acute symptoms about three years ago. Intravenous medication with salvarsan was refused by each. Wassermanns were negative. None of these patients gave the co-operation which is so necessary in clearing up this infection. As soon as the discomfort was relieved and the tissue looked fairly normal they stopped treatment. Needless to say, the organisms have never been entirely eradicated and each patient has occasional exacerbations. The tissues in the mouths are always considerably inflamed. Spirilla and fusiform are always present in fairly large numbers, especially in the interproximal spaces where the gum usually has a slight gangrenous surface. In three of these cases the alveolar process has been destroyed to such a degree that I have advised the extraction of all of the teeth.

These cases are interesting from another standpoint. Each of the patients is considerably below par physically without having any ailment other than that of Vincent's. After a few applications of salvarsan and removal of the gangrenous tissue there is marked improvement in their physical condition, but soon after discontinuing treatment the vitality again becomes lowered.

I am not citing these cases as conclusive evidence of the effect of chronic Vincent's upon the general physical well being. I cannot prove that there is any connection whatever, although I am convinced that each of these patients would be in much better health if the tissues in their mouths were not undergoing degeneration as a result of the large numbers of fusiform and spirillum of Vincent which are present.

Again, when it is realized that saliva is constantly bathing these gangrenous tissues and necessarily carrying portions of it, with such toxins and ptomains as may be formed during the destructive process, into the stomach of the patient, is it to be wondered at if resistance and vitality are greatly lowered?

Several writers have stated that Vincent's disease is only mildly communicable. I cannot agree with this, for I know of a number of instances where several members of families have been victims of the disease; one instance of a party of young people who indulged in kissing games, four of the six girls present later presenting for treatment; numerous cases of engaged couples, both of whom were affected; an institution for children in Newark, where a few years ago I had 28 cases, all under treatment at the same time; a college fraternity where all the boys in the house who used a common drinking glass were afflicted. Several who did not use this glass did not contract the disease.

From my observations, I would say that young adults are most often affected and that the region of the lower third molars is the most frequent starting place. Perhaps an explanation for this is the difficulty of properly cleansing the area, and the loose flap of gum tissue which is apt to partly overlie these teeth and so become traumatized during mastication. This produces lowered resistance of the part, as well as a break in the continuity of the tissues, which I believe is, to a very great degree necessary, for the organisms of Vincent to gain entrance, develop and produce the typical manifestations of the acute form of the disease. Food pockets between the teeth, where shreds of meat are allowed to collect and disintegrate, are ideal starting places for the infection.

Davis and Pilot, in the *Jour. A. M. A.* for Sept. 16, 1922, have pointed out that the crypts of the palatine tonsils are one of the chief habitats of B. fusiformis and spirochetes and from here they may spread to other points of the body.

It is possible that the organisms may be harbored in mouths without producing Vincent's disease. They may remain for considerable periods, until such time as conditions are right in the host for their entrance into the tissues, when rapid proliferation occurs. Kissing or other direct contact, as the common drinking cup, seem to be the most frequent modes of transmitting the infecting organisms. The degree of virulency of the organisms undoubtedly varies; Davis and Pilot have noted this, as have others. The resistance of individuals to the infection also differs.

It has become my practice to look upon any inflammation about the gingival margins, and also upon other lesions of the mouth, which are not unmistakably something else, as a possible Vincent's infection, and to make smears from the involved area before ruling it out. The acute ulcerating type is easily diagnosed; the characteristic gangrenous lesion, the raw bleeding surfaces when the dirty, grayish membrane is wiped off, the pain, and last but by no means least, the typical odor, all serve to identify it. Even in these cases smears are made for confirmation of the diagnosis and for observing the number and kinds of other organisms which are aiding and abetting the Vincent's infection. One point of importance in making smears is to get the material from the deeper portions of the lesions. I have on a number of occasions failed to find the fusiform bacilli and spirochetes in the superficial debris, only to find them in profusion by going deeper into the pockets with the loop.

The first symptoms noticed by an observing patient is an uncomfortable feeling about the teeth, or pain of an indefinite nature, accompanied by slight bleeding of the gums. If the point of infection is well back in the mouth, the throat is usually somewhat irritated and sore. The doctor and the patient are indeed fortunate if the diagnosis is made and the treatment begun at this point.

Ulceration is the next step in the progress of the disease. The ulcers may be confined to a single area or may be found in several parts of the mouth, on the gums, tonsils, fauces, pharynx, cheeks and tongue. If the lesions are about the teeth or jaws the bone becomes exposed and denuded and necrosis follows, at times with almost incredible rapidity.

Before outlining the method of treatment which I have found to give the best results, let me briefly touch upon some that have been advocated, which I have tried, and rejected for the reasons stated.

Proprietary mouth washes, several of which are highly commended by the manufacturers for the treatment of Vincent's disease, are absolutely without value. Peroxid of hydrogen often does more harm than good, acting as an irritant; although I sometimes use it in a spray to wash off the gangrenous membrane. Dilutions of the analin dyes are of value, but their use is objectionable because of the staining of the lips and teeth. The dye will penetrate cracks in tooth enamel, producing a permanent discoloration of the tooth. It sinks into the enamel at filling margins and goes deeply into the substance of synthetic porcelain fillings. I used a solution of gentian violet on one case in which there were many of these fillings in the anterior teeth; 20 to be exact. The dye penetrated the fillings to such a depth that all had to be removed and replaced with new ones. Occurrences of this kind are not conducive to that friendly feeling which should prevail between patient and doctor.

Chromic acid and trichloracetic acid are both irritants and caustics and when used on the soft tissues produce additional channels of entrance for the invading organisms. The same is true to a lesser degree of frequent applications of the tincture of iodin, a favorite remedy with some operators. Solutions of silver nitrate should never be used, for it is not efficient, and because it will stain the teeth, generally penertating to such a depth as to produce a permanent discoloration. Mercurochrome I have tried, but find it to be of no particular value in combating Vincent's infections. Direct application of ultra-violet rays proved to be very disappointing. To properly apply the quartz applicators in the mouth of a patient who has Vincent's disease is very difficult; at time quite impossible. The pain of making the application is hardly permissible even if good results were obtained. If the applicator is kept in one position just a few seconds too long—and tissues differ in their susceptibility to the rays—a burn results, which, as in the case with caustics, simply aids in spreading the infection.

The above are only a few of the agents which have been advocated for use in the treatment of Vincent's disease. I have tried these and many others, none of them giving outstanding good results, and a number proving to be very objectionable for one reason or another. I wish to state emphatically that I am not wedded to any particular drug or method of treatment; anything which will rid the patient of the infection in the most thorough manner, in the shortest

time, has my approval. Satisfactory results are what we desire, and with your indulgence I will tell something of the method which, with Nature's assistance, has been found most efficacious in my practice.

A patient presents for treatment of a condition, which from the clinical symptoms is diagnosed as Vincent's disease. From the deeper portions of the lesions, smears are made, stained with aqueous fuchsin or gentian violet and examined with the microscope. Large numbers of B. fusiformis and spirochetes are found. The whole mouth is then examined and all points of ulceration and inflammation carefully noted. It is then thoroughly washed with a compressed air spray of warm water, to which has been added tincture of green soap, in the proportion of ½ drachm to 4 ounces of water; or peroxid of hydrogen and water in equal parts. This is followed by several sprayings and rinsings with plain warm water. The ulcers and all inflamed areas are dried and then coated with salvarsan, the powder being applied directly to the surfaces and carefully worked into the spaces between the teeth, into all pockets and under the free margin of the gums. Some form of delicate instrument is required for this purpose and a slightly curved explorer which has been flattened sidewise answers the purpose admirably. Dampen the tip of the instrument, dip it in the powder, and that which adheres is carried to the desired point and applied as directed above.

It is necessary to remove as much of the slough as is possible before applying the salvarsan, because the organism which it is most essential to reach are those in contact with the vital tissue. At the same time great care must be exercised not to produce injuries which will allow B. fusiformis and the spirochetes to gain entrance to the uninfected tissues. The patient is given a 2 per cent solution of copper sulphate in distilled water, with instructions to apply it to the affected parts every two or three hours. Recently I have been instructing patients to use perborate of soda in place of the copper sulphate for home treatment, after the acute symptoms have subsided. I instruct them to apply the powder to the gingival margins and interproximal spaces several times daily with a flat-end toothpick. Care must be taken not to produce any injury. Results have been very gratifying. This drug, however, sometimes acts as an irritant and its use must be curtailed or discontinued when this occurs.

Arrangements are then made for an early intravenous injection of salvarsan, or an intramuscular injection of sulpharspenamin. Four to six decigrams of salvarsan is the amount generally administered. The patient is instructed to refrain from kissing, to use his or her

own drinking glass, and to refrain from smoking. Drinking at public places, as soda fountains, is prohibited because of the possibility of spreading the infection to others. It is also advisable to caution against putting the fingers into the mouth, for by so doing the organisms may be carried to some other mucous membrane of the body with unpleasant and disastrous consequences.

One complete mouth application usually requires 1 decigram of salvarsan. As the drug rapidly disintegrates on exposure to the air, only the quantity to be used on each individual case should be prepared. Salvarsan has been found to give better results than neosalvarsan.

To return to our patient; on the second day the treatment of the mouth is just the same in every particular as on the first visit. The third day the treatment is also the same, except that powdered copper sulphate is used in place of the salvarsan. These daily treatments continue for 10 days, smears are always made, all parts of the mouth are cleansed with the soap spray, and salvarsan and copper sulphate are applied on alternate days. By this time the mouth should be nearly free of Vincent's organisms; if, however, this is not the case, another intravenous injection of salvarsan is in order. Only in rare instances is the second injection necessary. The earlier treatment is started after infection has taken place the better will be the result. Generally the first treatment as outlined above, the removal of the slough and the salvarsan application, will give marked relief and most of the pain will subside within 12 hours. Solutions of salvarsan in glucose, glycerin or water, while of some benefit, are not so effective as direct applications of the salvarsan powder.

Some writers object to the intravenous administration of salvarsan. Reichman, in the *Jour. of the American Dental Association* for December, 1926, says it is "an irrational and empirically nonsensical procedure". I disagree entirely with him on this point. Since employing intravenous medication in conjunction with the local treatment, cases are now cleared up in about one-quarter the time it took when local treatment alone was depended upon. Investigations have shown that B. fusiformis and S. Vincenti find their way into the deeper tissues. Surface cleansing and the topical application of medicants is not sufficient to eradicate them, especially when lodged in deep pockets between the teeth, or in the crypts of the tonsils. Salvarsan intravenously provides a means of destroying those beyond the reach of direct contact with the drug. It can be administered with very little danger or discomfort to the patient. The above

appeal to me as being sound arguments favoring this therapeutic measure.

As smears begin to show fewer organisms, we search for the points where they are breeding. Oftentimes a smear from the upper jaw will be negative and one from the lower positive, or vice versa.

We divide the mouth into four quarters, two upper and two lower, and make smears from each, going about every tooth in the sector for material. Any section in which the organisms are found is further examined by making separate smears from about individual teeth. As a means of identifying areas we use either a diamond or wax pencil to mark the slide. By following this technique, it is possible to determine definitely the focus of infection, and efforts to eradicate the organisms can be concentrated at that point. This may seem like a great deal of unnecessary labor, but it is impossible to tell by the appearance of the tissues when they are free of the infection.

The assertion has been frequently made that Vincent's disease yields without difficulty to local treatment in from a few days to a week. It is absolutely true that the ulcers can be healed and the pain relieved in a few days, but it is just as true that the organisms will still be found present in the mouth or throat in large numbers after the tissues have taken on a reasonably healthy appearance. It is also true that unless the organisms are entirely eradicated at this time, or if an infected pocket escapes notice, the patient is very apt to have another acute attack of the disease. This statement must be qualified to a degree; sometimes B. fusiformis and the spirochetes become less virulent in the host and produce a low grade chronic type of infection, which is often classified as pyorrhea.

To again return to our patient; the tissues will have taken on a reasonably healthy appearance in 10 days or so and this, to my mind, is a very important time, for unless the patient is sufficiently impressed with the necessity for continued treatment he or she is apt to stop.

The intervals between visits may now be gradually lengthened to two or three days, provided the patient is intelligently and conscientiously co-operating.

As soon as it is possible to do so without producing injury to the soft tissues, all accumulations of tartar about the teeth should be removed, and all other points of irritation must be done away with, for every such irritated area affords a lodging place for Vincent's spirillum and B. fusiformis. Extracting of teeth during the active acute process of the disease is only resorted to if the necessity is absolute. Scrupulous cleansing of the mouth is of very decided

advantage, in fact it is imperative, and I have found that a few drops of tincture of green soap on the toothbrush, particularly after meals and at bed time, will materially aid in removing all food and other debris. Of course the patient must endeavor to reach all parts of the mouth during the brushing process. Perborate of soda used on the toothbrush, as a tooth powder would ordinarily be employed, is of real value and many patients prefer it to soap. A solution of this drug may also be used to advantage as a gargle, two teaspoonsful to a glass of water. Tooth paste or powder is not allowed, as we do not care to risk the irritation which might follow the use of either.

Copper sulphate may produce a greenish stain on the teeth. This is only on the surface and can be easily removed when the teeth are thoroughly cleaned and polished, as must be done before the case is dismissed. I have patients report at weekly intervals for two months after the smears from the entire mouth have been negative on three consecutive visits. Usually they are perfectly willing to do so, for one experience with an ulcerative Vincent's infection is generally enough to satisfy almost anyone.

The use of salvarsan and copper sulphate is now discontinued, unless a smear should be found positive, and we depend on thorough cleansing with soap or perborate of soda, advising their continued use for at least three months.

Massage of the gums with the fingers is also advocated after the mouth has been found to be free from Vincent's organisms. This aids in restoring a healthy tone to the tissues by increasing the circulation.

Modifications of the treatment herein advocated will have to be made to suit individual cases, no two of which are exactly alike. However, as a working basis it will, if carefully applied, give results more quickly and more certainly than any with which I am familiar.

To prove that it is possible to entirely rid the mouth of a patient who has had Vincent's disease of the B. fusiformis and the spirochetes, I have made it a practice to proceed as follows: whenever a patient who has had Vincent's comes to the office I take material from all parts of the mouth for smears, and I can report that in a large majority of the cases that have been discharged as cured, there has been no recurrence and none of the organisms of Vincent are to be found. In some instances I have made examinations of this kind over periods up to five years after the patient was discharged. The above refers particularly to the acute cases, in which it is possible to institute treatment fairly early in the course of the disease and where tissue destruction has not been too great. When treatment

has not been thorough, and when the loss of gingival and alveolar tissue has been severe, the organisms find their way into the deeper structures, and I question if they are ever entirely eradicated from about the teeth and gums until the teeth are all removed.

CONCLUSIONS.

Vincent's disease is of sufficient importance to warrant careful consideration and thorough treatment by dentists and physicians and the co-operation of members of these professions is essential to properly combat it.

It is very prevalent, and the necessity for early recognition and thorough treatment is imperative.

When B. fusiformis and Vincent's spirochetes are present, large numbers of cocci are found associated with them, and the effect of the combination on the human economy is certainly far from beneficial. This fact should be given due consideration by the internist.

The presence of large numbers of the organisms of Vincent in the mouth is unquestionably a factor in the production of irritation and infection of the structures with which the nose and throat specialist is particularly concerned.

Microscopic examination of smears from infected areas is the only safe means of determining the effect of treatments.

In closing, let me again impress upon you the necessity for early recognition, early and thorough treatment and also that the progress of the treatment must be followed with microscopic examinations of smears taken from all parts of the mouth.

31-33 Lincoln Park.

A SIMPLE AND SAFE OPERATIVE TECHNIQUE ON THE NOSE UNDER GENERAL ANESTHESIA.

DR. S. B. FORBES, Tampa, Fla.

For several years I have considered reporting my own experience in nasal work under general anesthesia.

In discussing this with other men the majority seem to feel that operative work on the nose under general anesthesia is a thing that can be accomplished only with great difficulty.

This view is not correct in any respect. I have been using the method which is herewith described in very many cases for the past seven years and I have never experienced the slightest difficulty in any of the various types of nasal operations.

Any method of anesthesia may be used to carry the patient to the third stage. We then insert the Crowe-Davis mouth gag and the ether vapor is administered through this. The mouth is about one-half open. Next a postnasal plug is inserted in the usual way and the anesthetist is instructed to keep the nasal string of the plug taut and to keep a close watch on the pharynx for any blood that might get by.

A capable anesthetist will soon learn to take care of the throat by having the suction constantly at hand. However, the operator should at frequent intervals throw the light from his head mirror into the throat to see that it is dry.

After the usual skin preparation long strips of gauze are soaked in adrenalin, the excess squeezed out and then these are packed into the nostrils, being sure that they are well back to the choanae on each side and packed from the floor to the roof. These packs should remain in at least five minutes before any work is started. The patient's head is then elevated to about a 45° angle.

After the packs are removed you will find as little blood in the operative field as under most local anesthetics.

You can now go ahead and do a submucous resection, ethmoidal exanteration, intranasal frontal, maxillary or whatnot.

For sphenoidal work the plug must be pulled back into the mouth while the face of the sphenoids are removed.

In this work it is important to have the suction in the mouth to take care of the bleeding. At the completion of the sphenoidal work the plug must be pulled back to the nasopharynx.

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I am going to digress slightly in reference to the radical maxillary operation of the Caldwell-Luc or Denker type.

Here again you use the Crowe-Davis gag. If you are going to work in the right side use the gag with the left arm, if the work is done on the left side use the gag with the right arm. In this way it will not interfere with the operative field and at the same time you have absolute control of the patient's condition, as you have a wonderful exposure of the pharynx.

In the radical antrum work the head of the patient is not elevated quite as high as in the intranasal operations, but it is much easier to work in a slightly elevated position than in the horizontal.

I wish here to make another digression in reference to the Crowe-Davis gag. Of all retractors for radical maxillary work, my choice is the plain blade of this instrument. It works perfectly in retraction of the upper lip and better in the alveolar incision. An operator is at a loss in this region without perfect retraction.

Returning to the true subject, place your packs in the nose as under local anesthesia, being careful that you have a complete pack so that there is no hemorrhage while the patient is reacting from the anesthetic. After the packs are in place I frequently watch the throat for any bleeding for several minutes after anesthesia is stopped. If there is any question I let the patient remain on the table with the gag in place and keep the suction close at hand until he has partially reacted.

The head of the bed is elevated and ice compresses placed on nose early. The postnasal plugs remain in place about 12 hours and the intranasal packs from 24 to 36 hours. Antral packs are removed on the fourth post-operative day.

My observation has been that patients on which the work has been done under general anesthesia bleed less when packs are removed than those done under local.

In closing, may I state that I do not wish to be placed in the position of advocating general anesthesia for all nasal work, but we all have certain patients, particularly children, where local anesthesia cannot be used.

409 Citizens Bank Building.

CASE OF NEUROSIS DUE TO TOO MUCH OPERATIVE WORK ON THE NOSE.*

DR. WOLFF FREUDENTHAL, New York City.

About 14 years ago, this patient consulted me, giving the following history: For some difficulty in breathing he had been referred to a rhinologist of this city, who removed the middle turbinated on one side and afterwards treated him for several months without any apparent relief. He then consulted another rhinologist, who removed the middle turbinated of the other side, and also treated him for several months. The result was the same, that is nil. A third rhinologist took off the anterior tips of both inferior turbinated bodies and operated on the septum. Finally the patient landed at my office.

On examination, the nares appeared in almost an atrophic condition. The mucous membranes were pale and covered with crusts, but not enough to interfere with normal breathing; yet the patient told me: "Since my first operation, I feel no air. Plenty of room in the nose." Here we have one of those cases which I described about ten years ago, in which a destruction of the physiological function of the nose resulted after operations on the nose (*New York Medical Jour.*, Nov. 9, 1918).

I treated the patient during this time, on and off, mainly along hygienic lines. He changed his residence for better quarters, went sea-bathing all summer, and spent a good deal of the time in the mountains during the winter. The result is that today, after 14 years, the nasal passages have narrowed down considerably, transudation of lymph and blood takes place within the mucous membranes so that they appear reddish and fairly normal. In spite of everything the patient still keeps on telling me, "I feel no air in my nose".

What has happened in the meantime is that a neurotic condition has been established so that in spite of the improvement in the nose he still sticks to his early statement. Other neurotic conditions have developed since in different parts of the body, so that he cannot take any coffee, because his "abdomen swells inmmediately". When I asked him about tea, he replied that that was worse still. The patient has been under treatment by several stomach specialists; he also was under the care of several neurologists and rhinologists. One

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of the latter advised him, "Young man, don't throw away your money; forget your nose and your nerves, and you will be well". That sounds very well and honest, but I don't agree with it. This patient could not forget his ailments, in spite of the doctor's advice, and was ready to go to some charlatan, when he consulted another physician, who referred him back to me.

Let me give an example of a case that occurred to me several years ago. One of the very rich men of New York City consulted me on account of a mild nasal catarrh. He was one of those men who is never satisfied with anything done for him. He bothered me, so to say, day and night. Whenever he came to my office, he felt worse, no matter what I tried to do. Finally, one morning he asked what I thought of Christian Science. I told him, "That's just the thing you ought to try". Two years later I met him on the street, and remarked how well he looked. He replied, "Doctor, I owe that all to you. Indeed, I am eternally grateful to you for what you have done to me. I shall never forget that Saturday morning when you advised me to go to the Christian Scientists. Ever since then I am absolutely cured".

What happened to the patient is this: They made him give up his business, move to the country, and kept him out in the open air as much as possible, which was sufficient to cure his slight ailment. He would not do that for me, although I urged him.

Quacks only too often avail themselves of the opportunities that we throw away. This is by no means a question of finances, but of a very important part of medical practice. We, as physicians, are here not only to cure physical ailments, but we have to change the mental attitude of psychogenetic patients and establish their mental equilibrium. If we don't know how to do that, we should go out and learn.

This case shows just the reverse of what we have been accustomed to see and do. Formerly, we tried—as in bronchial asthma—to cure the nervous condition by removing polypi from the nose, and succeeded in a number of cases. In this instance, a neurotic condition developed after the patient had been operated upon—a consequence which can and should be avoided.

24 West 88th Street.

PERICARDIAL ESOPHAGEAL FISTULA, FOLLOWING FOREIGN BODY IN THE ESOPHAGUS. PNEUMOPERICARDIUM; AUTOPSY REPORT.*

DR. CHARLES J. IMPERATORI, New York City.

G. P., female, age 13 months, admitted to the New York Post-Graduate Hospital, July 22, 1927, Chart No. 26707, with a diagnosis of bronchopneumonia. The child had been ill for over a month and was in very bad physical condition when seen.

It was the opinion of the attending physician, who referred the patient to the hospital that she would very likely die because of the bronchopneumonia. An X-ray taken at that time revealed a foreign body, that is, an open safety pin at the cardiac end of the esophagus, a bronchopneumonia and a pneumopericardium. The dimensions of the pin were as follows: The spread or plane of expansion was nearly 50 m.m., or 1⅞ inches. The keeper branch, including keeper, to clamp, 35 m.m. The pointed branch, 30 m.m.

The last finding, that is, the pneumopericardium, was not reported by the radiographer. A study of the location of the point of the pin showed that it had perforated the esophagus. Because of the child's condition preceding the operation, a time limit was set of not over five minutes' duration of operating time. No anesthesia was used. Passing a small esophagoscope, the pin was extracted within the time limit. As is the usual custom in foreign body work, a post-operative X-ray was taken and this showed no further foreign body, but exactly the same pathology as the original radiograph. However, on reading the plates at this time the radiographer reported that there was a pneumopericardium. On further examination of the original plates, pneumopericardium was found. The pathological findings at the site of the foreign body were as follows: The point of the pin had penetrated the esophageal wall to the spring and clamp and there was an area of about 3 m.m. in diameter of granulations around the shaft of the pin.

Attempts to dislodge the pin and push it further down into the esophagus so that the point could be grasped were unsuccessful, and knowing that it was a soft brass one, the following method was

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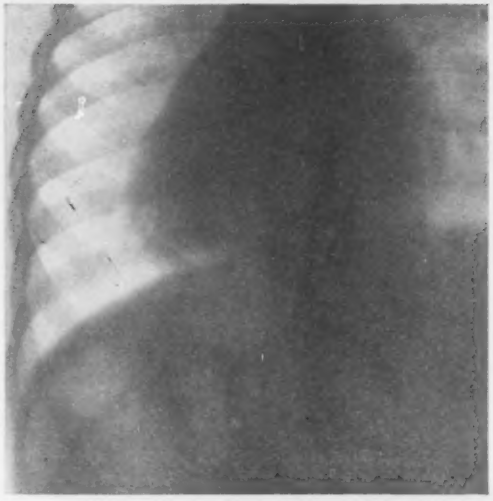

Fig. 1. X-ray showing safety pin in lower esophagus with air in the pericardial sac. This does not show the degree of expansion of the pin, which was 50 m.m.

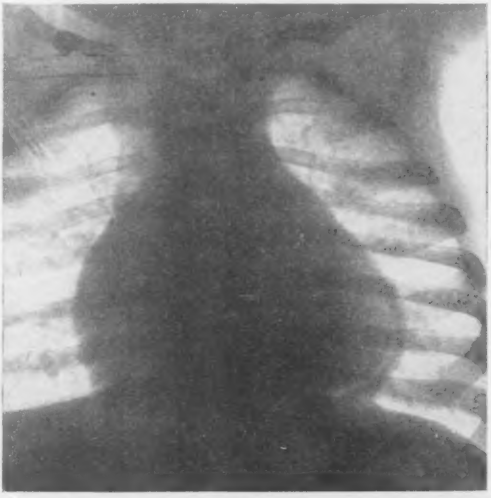

Fig. 2. X-ray taken 24 hours after removal of pin, showing pneumopericardium on opposite side.

used: This method of removal is that suggested by Jackson, of straightening the pin against the tube mouth with the keeper and keeper branch within the tube; by pushing the tube over the keeper and keeper branch—the pin being held stationary in the tube and a lateral movement of the tube away from the point of the pin is done

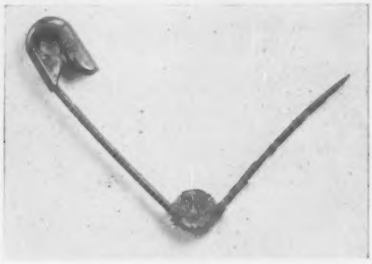

Fig. 3. Safety pin.

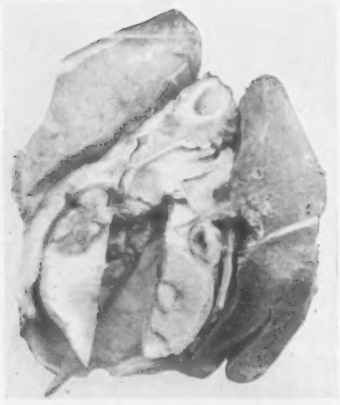

Fig. 4. Anterior view of heart and lungs. Heart muscle is cut away to show pericardium. Both the visceral and parietal pericardium are very much thickened. There is a wooden stick in the fistula. There is considerable thickening of the pericardium and two abscesses of the myocardium. The lungs show bronchopneumonia.

after the tube is pushed down to the spring, the pin then being further drawn into the tube. During the operation the child was in fair condition and was not asphyxiated to any degree.

Postoperative Notes: The child was able to eat much better, temperature dropped to 99°, and generally seemed to be very much

improved. However, the bronchopneumonia increased but Roentgenograms showed no pneumopericardium. Seven days after the operation the child died. The clinical diagnosis was bronchopneumonia.

Autopsy Report: There was a pericardial esophageal fistula, bronchopneumonia with infarct, left hydrothorax, chronic purulent pericarditis, terminal sepsis. The probe passed from the esophagus into the pericardium near the entrance of the right vena cave.

This case is of special interest from several standpoints. The mother knew of no accidental swallowing of the safety pin and was greatly surprised to know of our findings. This woman was of moderate intelligence, who took care of this child herself, and insofar as she recalled, no safety pins were missing from the child's clothes.

Fig. 5. Posterior view of heart and lungs. The esophagus is split and deflected to either side and shows a wooden stick in the fistula. Lungs show bronchopneumonia.

How it got into the child was a mystery to her. Naturally, the mother felt very much perturbed that this condition existed, without her physician, who had been treating the child for about a month before admission to the hospital, knowing of the existence of the foreign body.

Recently I have seen two children with safety pins in their esophagus, and both doctor's children. One was treated for retropharyngeal abscess, wry neck and cervical adenitis, and the other for indigestion. Radiographs showed a safety pin in each esophagus and both were successfully removed. The first patient complained for over a month and the location of the pin was in the epipharynx, and the

second patient gave symptoms for three weeks. Both parents of these children knew of no lost or misplaced safety pins. In the first case, repeated Roentgenograms had been taken but the radiographs never included the pharynx. In the other case the mother, after a period of medical treatment, had insisted on a radiograph, that revealed the pin.

Also, it is of passing interest to know that a great many of the wild animals in the Bronx Zoo die from pneumopericardium, which is induced by their biting off and swallowing pieces of barbed wire of the fences of their enclosures, and thus injuring their esophagus.

In children a routine chest examination should be confirmed by Roentgenograms and to be complete should include the occiput and tuberischii. Jackson has postulated that all children and infants suffering from any symptoms whatsoever should have Roentgenograms taken. This dictum, had it been followed, would very likely have saved the life of this child, for it is unusual that a pin perforates in the early period of its stay in the esophagus, and rarely would such a size pin be lodged at the cardiac end of the esophagus in a child of this size and age. Had the pin been discovered, it would have been more easily removed than later.

The frequent and unnecessary discussion both by physician and patient, leading up to the taking of a Roentgenogram, tends to confusion. A Roentgenogram may be taken of the neck, chest and body of an infant or child with considerable ease. As with all laboratory methods, the interpretation must be carefully considered, either from a negative or positive standpoint by the clinician before any operative measures are instituted.

17 East 38th Street.

DYSPHONIA SPASTICA (Spastic Hoarseness).*

DR. L. GLUSHAK, New York City.

The functional disturbances of the larynx are manifested through a change in the performance of its subserving actions in the processes of respiration and voice production. Placed at the entrance of the trachea, the larynx through its motor mechanism, protects the lower respiratory passages against the entrance of noxious particles, and exercises a controlling influence on the column of exhaled air, throwing it into vibration, and thereby bringing forth the act of phonation. It is a highly specialized physiologic function, and as such is subject to control by the will.

Voice is the result of a complexity of co-ordinated actions, commencing with respiration, followed by phonation in the larynx, resonation in the upper air passages, and ending with modification and emission in the oral cavity. Modern students of the practical singing and speaking voice accredit the larynx with only a small role in this complicated act, premising that it only produces the simple, unamplified tone, or crude element, which is to be further polished and enriched by the overtones in the resonating cavities, *viz.*, the pharynx, nasopharynx and nasal passages, until the desired effect in the particular quality of tone is attained. This, however, cannot be entirely conceded, as the larynx is not merely a reflex sound-producing organ, but at the instance of the will is capable of modifying the sounds into tones of varying character, such as whisper, singing, speaking, falsetto and whistling.

In the advance of physiological evolution, phonation has been a newer addition, and subserves not only a facility of interindividual communication, but by its more polished and cultivated form in the human, has become a means of transmitting our innermost emotions in a most impressive and expressive way, either through speech or song. Accordingly, the existence of a cortical centre for phonation has been demonstrated experimentally by Krause, Horseley, Semon and others. It is bilaterally situated, and each side innervates both adductor muscles in the larynx so that stimulation of the one centre causes an approximation of both vocal cords. These experiments have also shown that if the irritation of this centre be continued,

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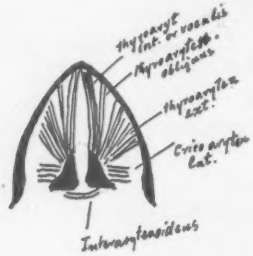
the vocal cords are thrown into a spasm which may result in a tetanic closure of the glottis. The glottic function is thus seen to be an integer in the proper provocation of the voice and speech, under the guidance of an independent cortical centre, co-ordinating with respiration, resonation and associated mechanism of the mouth, tongue, etc.

The tone-producing apparatus consists of two fleshy prismatic processes, projecting from the internal surfaces of the thyroid and arytenoid cartilages, each being made up principally of a composite fan-shaped muscle, the adductor or thyrocrico arytenoideus, which with the interarytenoideus approximates the vocal cords and arytenoids, narrowing the gap between them where the column of air is set in vibration. This adductor, or sphincter laryngis, as it is sometimes called, through its component bundles acting in parts or altogether, produces the various modifications in phonation.



In the first step of tone production, the cords and arytenoids are approximated by the cricoarytenoideus lateralis bundle together with the interarytenoideus. When the latter is left out, the arytenoids remain separated and, with the escape of air between these while the cords are in the act of vibration, a whisper is brought forth. After the glottis has been narrowed, the vocal cords are further approximated by the thyroarytenoideus externus. The tone is produced by changes in the internal tension of the vocal process acting on its differentiated part the ligamentum vocale, forming the edges of the glottis, through the action of the thyroarytenoideus internus, or vocalis muscle, either as a whole or through its component bundles, to evoke various forms of tone, *e. g.*, the various registers, the piano, forte, crescendo, falsetto, as well as determining the pitch, *i. e.*, height or depth of tone. Under volitional impulse these changes are brought about; and thus the laryngeal mechanism is adjusted to

respond in diverse forms of tone, under the censorship of the ear, acting both as critic and guide.

Aberrations from the normal actions of the muscles of phonation will result in changes of form of tone, ranging from the slightest defects in voices of highly cultured artists, to grave impediments in producing audible sound for practical everyday speech, down to complete loss of voice.

Omitting the organic, obstructive, paralytic and articular factors in laryngeal pathology, the functional neuroses of the larynx form a large and frequent group, ranging all the way from the asthenic conditions of the voice, as hysterical aphonia, phonasthenia or weakness of the voice, fatigue of voice, to the spasmodic affections, which manifest themselves either as respiratory disturbances or phonetic disco-ordinations.

As an example of respiratory glottic spasm, we have the condition known as laryngismus stridulus occurring in very emotional children disposed to rickets and malnutrition, who in a fit of excitement, after a few stridulous inspirations, fall into a characteristic convulsive state in which respiration ceases. In adults, respiratory spasms occur as a functional impairment, reflexly provoked by irritation of the recurrent laryngeal nerves due to intrathoracic pressure, or central vagus lesions, as in laryngeal crises in tabes, direct irritation of larynx by subglottic or glottic neoplasms. The nervous cough is another paroxysmal glottic condition of unknown origin. It was named "the barking cough of puberty," occurring in the teens of both sexes. These patients are in no way affected as to their general health, while their affliction, which curiously prevails only during the day, causes them to be of great annoyance to their neighbors.

Of an outstanding type of neurosis is a peculiar form of hoarseness known as dysphonia spastica, in which, as the name implies, a spasm of the adductors is provoked on attempt to phonate producing under great stress, a toneless, almost whispered voice, with which, on sustained effort to continue, a high pitched whistle or screech intermingles and sometimes becomes so aggravated that the patient ceases to talk. It is a cramp of the glottis, involving all the components of the adductors, and especially marked in the thyroarytenoideus internus or vocalis, the innermost bundle, which has been observed to overlap the opposing vocalis on phonation, in the severe forms of this disease. Associated with this action, we sometimes find that the false cords are approximated by the intense inward pull

of the adductors; even the epiglottis is brought closer into the larynx through the action of some muscle bundles running into the aryepiglottic folds. Laryngoscopically, no gross anatomic change is seen, although if the condition continues for a considerable period, a laxity or hypertrophy of the mucosa of the false cords is liable to set in. The respiratory discrepancy in the picture plays a minor role, except in the development of the trouble; for, a normal, quiet and co-ordinated respiratory mechanism is absolutely necessary to give the cords the necessary poise and regularity of action; and so we find in stuttering a deviation from the normal respiratory act throwing the phonatory and enunciatory parts of speech production out of gear. Some have considered this spastic dysphonia as a form of stuttering of the larynx, and in this conception we find a clue to the diagnosis and therapy.

The condition develops on a neurotic basis in a highly-strung adult, usually a professional voice-user, as a public speaker, when as a result of vocal strain, a fatigue of voice appears as a result of improper use, or not resting the cords sufficiently after an acute laryngitis. Under such circumstances the cords begin to fail in their proper approximation, and resemble an internus paresis. A further attempt to speak is made to overcome the initial disability. This time a harsh sound is evoked, displeasing to the patient's ear, and in the attempt to improve the tone to a more effective form, it is further forced. The patient becomes physically depressed over the situation and precipitates in a perverted habit of false phonation in which the spasm of the glottis is established, even including the respiratory part of phonation, as the diaphragm and chest expiratory muscles. The patient gets red in the face and presents a very tense appearance of grave concern over his plight, and is soon compelled to give up the attempt to speak in the endeavor to make himself audible to others. The condition varies both in severity as well as mode of development. It may manifest itself suddenly as a pure neurosis following traumatic shock, or as a functional nerve disorder associated with hysteria or neurasthenia.

The writer wishes to report the following case that came to the Post-Graduate Hospital clinic. E. B., age 39 years, housewife, complained of loss of voice, two months' duration, coming on rather rapidly, giving as a cause a slight cold. She is a low-statured, well-nourished woman, rather quiet, and of secretive demeanor. Her physical status, including a chest and blood examination, is satisfactory. The upper respiratory passages, including sinus, show no pathological process. Her voice in the endeavor to speak is intensely

forced and with the words only a toneless sound is emitted, intermingled with a screech in her endeavor to make herself heard. She gets red in the face and, not succeeding to carry on a conversation, desists from speaking, in a disheartened mood. On further questioning she informs us that she had no nose and throat ailments and is slightly disposed to infrequent attacks of colds. Her social environments are of great importance in the etiology of her present ailment. She is the mother of a large family, and lives with her husband, who is an habitual drunkard and violently disturbs the family life, both by failing to provide adequately, and boisterously attacking his wife and children, sometimes even threatening to kill them. Under such circumstances, quarreling and shouting is constant and threats and fright have often thrown her into a hysterical fit of what she calls "shivering when her voice stopped on her." From this point her spastic hoarseness started and thus she fell into the habit of speaking on a very high-pitched tone which after a few words became a forced noise. Laryngoscopically she shows an approximation of the false cords, which are a little redder than usual, and hiding somewhat the true cords, and an intense forcing together of the latter, disabling the emission of any sound. Upon request to breathe, the cords separate and show themselves to be free from any outstanding pathology except for a slight irregularity in the edges. She recovered her voice after a short course in respiratory and phonetic exercises. There has been a slight recurrence on the occasion of one of her frays at home, but now that she has learned to assume the correct manner of phonation she is able to correct herself.

Upon the recognition of this condition rests the therapy, as one is apt frequently to label this disturbance chronic catarrhal laryngitis and employ various intralaryngeal medicaments, only to aggravate the condition, or advise rest, which would be of no avail. The patient must be shown the normal respiration of speech with the tone produced on the exhaled breath. He is to endeavor after an initial deep, noiseless inhalation to exhale slowly, evenly, and during this process to bring forth a low singing chest tone on a vowel of the speaking register, starting with "a" like in father, on "c" for women and an octave lower for men. Thus the patient's voice is placed on a new threshold of speaking tone. After a while he is given to read in syllables, singing one syllable into the other in a long drawnout monotone. These procedures tend to co-ordinate new respiration with proper phonation and introduces to his ear a new tone, which he soon learns to imitate and follow, and forgets his recently abandoned habit. It is surprising how quickly some are restored to normal. Stubborn

and long cases take considerable exercising, psychotherapy, and perhaps removal from existing environments.

Dysphonia spastica is not as rare a condition as was considered by the earlier observers. It is a spastic response of a highly specialized function to an intra or extra laryngeal irritation, in an individual with a neurotic predisposition, and may have its origin in a pathological lesion, such as laryngitis of a mild nature or slight growth, giving initiative to the irritation. Thus it may be of more frequent occurrence and the functional and pathological elements must be recognized to treat the condition successfully.

317 West 89th Street.

SUCTION APPARATUS WITH SPECIMEN COLLECTOR.*

DR. L. GLUSHAK, New York City.

The apparatus herewith presented is intended for use in the course of everyday practice of oto-rhino-laryngology, to remove and collect discharge for microscopical examination. It consists of the following parts:

a. Collecting bottle, graduated and easily removable from the cap which is attached to the *b.* suction tube. At the right end it receives *c.* tubes of various dimensions and lengths for use in nasal, pharyngeal, laryngeal cavities, ear canal and tympanic cavities. These tubes vary in diameter from 1-3 m.m.; they are malleable and can be bent in any shape. *d.* Opening for insertion of universial suction tip of any standard suction motor apparatus. *e.* Rubber ball with adapter to be used at the bedside in place of suction machine. *f.* Cap to cover collecting bottle to be sent to laboratory for culture.

The removal of discharge from any cavity for diagnostic or therapeutic purposes with cotton and applicators offers the following objections: *1.* It is painful to the patient. *2.* The discharge can hardly be reached in remote depths or around corners. *3.* The direction of the flow of the discharge cannot be ascertained under

Read before the New York Academy of Medicine, Dec. 28, 1927.

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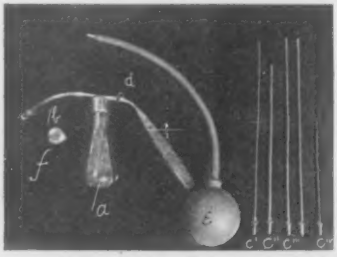
the direct supervision of observer's eye, as an applicator obstructs the examiner's vision. *4.* Smears for culture cannot be made accurately with removable applicators without contamination on withdrawal.

The apparatus presented has a bent arm which carries the various tubes for the different cavities under treatment. The chief feature



of the instrument is that it does not obstruct the vision of the examiner while he is concentrating on the part in question, especially if it be at a great distance, as in posterior sinuses, hypopharynx, larynx, etc. Discharge from the entrance to the Eustachian tube can be removed while observing it through the Nasoscope from the adjacent nasal passage.

317 West 89th Street.

A NEW RESPIRATORY SHIELD.

Dr. Simon Jesberg, Los Angeles.

I hereby present a shield which is used to prevent respiratory infections. The device consists of a transparent shield which is supported by two parallel wires. The upper portions of the wires pass through a tension spring box which holds the shield in position. The tension spring box allows the shield to be attached to any head-band having a projecting stud, without the aid of screws or special tools.

Besides protecting both the physician and the patient from droplet infection, this device allows the physician to continue the examination

of the oral field while the patient is coughing. The device is light in weight and can be quickly rotated out of the way for telephoning or conversation.

There are on the market several devices of this sort. For ten years I have used modifications of the type described above, but none of these have been as flexible as this. The shield in its present form (with some slight modifications) has been in use for two years. It was perfected for me by Mr. J. J. Cantor. whose advertisement appears on page 7 of this issue.

500 S. Lucas Avenue.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, Dec. 28, 1927.

Dysphonia Spastica. Dr. L. Glushak.
(Published in full in this issue.)

Suction Apparatus, with Specimen Collector. Dr. L. Glushak.
(Published in full in this issue.)

Pericardial Esophageal Fistula, Following Foreign Body in the Esophagus. Dr. Chas. J. Imperatori.
(Published in full in this issue.)

Vincent's Infection of the Mouth. Raymonde Adair Albray, D. D. S.
(Published in full in this issue.)

Systemic Manifestations of Vincent's Infection. Dr. Charles F. Tenney.
(Published in full in this issue.)

DISCUSSION.

DR. DWYER: First of all, I wish to congratulate the various speakers on the comprehensive manner in which they have treated the subject. I do not feel that much progress has been made in our knowledge of the subject during recent years. Basically, I have considered that the disease is nearly always a secondary process, not a primary one; that preceding the local lesions, there is a profound change in the chemistry of the body; that noma or phagedenic ulcer, stomatitis, and in some instances conjunctivitis or lesions of any mucous membrane are of this type. The organisms are often found in the normal mouth, under ordinary circumstances probably as harmless saprophytes, and if some change in the body occurs they then start up as parasites. I do not feel that the last word has been said on the bacteriology of the disease. Years ago I paralleled Tunicliffe's work, with complement fixations, etc., and got nowhere. The observations as to the identity of these organisms and the drawings from Leewanhoek are very interesting. Some years ago I had the pleasure of seeing Leewanhoek's drawings, and in my opinion they are identical.

In conclusion, I feel that the observations that have been presented serve to focus attention to the subject and bring the whole matter up to date.

DR. LORE: This has been a very fascinating subject to me for a number of years. In 1921 I presented a paper before the Association of Italian Physicians in America on infections of the ear, nose and throat by the organisms under discussion. Vincent's organism is not confined to the mucous membranes; it may attack the skin as well. At the New York Foundling Hospital we had a case of Vincent's angina of the stomach, terminating in death. The question of whether it may be endemic or epidemic: In an institution it may be endemic. I saw over 200 cases in all forms over a period of two years.

As far as Vincent's being a primary lesion, I have seen only one case, which I studied very carefully—a middle ear infection. The child had been perfectly well and suddenly complained of earache. An incision was made, and a thick, dark red fluid came out, very, very foul. A smear showed the fusiform bacilli and Vincent's spirilla. In this case we also recovered the organisms from the adenoid.

I would refer you to Halsted's classification of Vincent's angina (Trans. A. L. A., 1912), the diphtheritic and syphilitic types. The diphtheritic type usually occurs in children and may simulate diphtheria. Both conditions may coexist in the same patient. Any suspicious case should have not only a smear but a culture taken. If a case of diphtheria does not progress favorably a smear should be taken to determine the presence or absence of the Vincent organism.

The syphilitic type occurs in adults. I was surprised that the speakers did not advocate a routine Wassermann test. I have seen cases where Vincent's angina was superimposed upon a syphilitic lesion in the throat. These are the cases that improve rapidly under the administration of salvarsan. Nor does a negative Wassermann absolutely rule out syphilis. The clinical picture and history are all important. I have had six cases of chronic mastoid in which we found Vincent's organism. In these cases we did not feel that the lesion had been primary. We thought they were all secondary up to the time we got the acute ear; then we began to feel that they may have existed primarily.

No one has mentioned the laboratory experiments carried out by an investigator in regard to the use of salvarsan—salvarsan and its action in vitro. This worker came to the conclusion that salvarsan has no action on the organisms under discussion and he concludes that there is no basis as to why salvarsan should be of any use in the treatment of Vincent's angina. Personally, I feel that the improvement noted after the use of salvarsan locally is due to cleaning out the infected area and allowing air to reach it. In using it locally, you have cleansed the area and have exposed to air. It is an anerobic organism and the presence of air will retard its development.

The first speaker spoke about the use of caustics. One must not overuse caustics; they devitalize the tissue; personally, I favor the use of hydrogen peroxid; I have also used formaldehyde. That is not empirical. Formaldehyde is used on the basis that the tissue affected by Vincent's organism is very boggy—has an enormous blood supply and if you can tone up the tissue you will have a better field. Personally, I first cleanse the area very carefully with peroxid, exposing the ulcerated area, this is then dried and a mild antiseptic applied. The multiplicity of treatments is some index to the haphazard nature of the treatment.

At the New York Foundling Hospital, where Vincent's infections existed (endemically), we tried out various local treatments—one group was treated with sterile salin solution. The involved area was cleansed, exposing the ulcer to the area. These cases got well.

Another group was treated with hydrogen peroxid and mild caustics. This method gave the quickest results. Still another group had the diseased area treated with salvarsan—these cases also got well. Our impression was that the real improvement was due to air (oxygen) being able to reach the actual diseased area, and not to the use of any antiseptic. The action of sodium perborate is based on this fact.

In using formaldehyde locally in mouth lesions, my practice has been to: 1. Cocainize the diseased area; 2. clean away the debris by means of hydrogen peroxid; 3. dry the ulcerated area; 4. then apply sol, formaldehyde de 40 per cent, making sure, however, that the applicator does not contain an excessive amount of solution.

When the patient uses it at home, he is given a prescription for a 40 per cent solution of formaldehyde and then instructed to start with 20 drops in a half a glass of water and to use this as a mouth wash. About five drops more are added to the original dose daily up to the point of tolerance.

DR. JACOB SOBEL: As one, who with Dr. Charles Hermann was the first in this country to call the attention of the American medical profession to Vincent's angina, I may be permitted to say a word. I confess that in discussing the subject tonight I do so for sentimental and personal reasons and largely because of a desire to reminisce, realizing at the same time that in so doing I acknowledge to the fact that I am getting somewhat older. As I entered these rooms tonight a member of your Section said to me, "Sobel, you are out of your sphere". Perhaps he was right, for, after all, I am a pediatrician and not a rhino-laryngologist. On the other hand, perhaps he was wrong. Let us see. Today, Vincent's angina is commonly known and recognized, and interesting studies and observations have been made upon it from numerous angles and quarters by many physicians and bacteriologists, as evidenced by the excellent papers presented this evening. But 26 years ago little was known or heard of this subject in our country, at least, and when I ask for the privilege of the floor tonight I do not feel that I should offer any special apology, for to receive this privilege once every 26 years it not requesting too much. I,

therefore, take the liberty and privilege to show you, Mr. Chairman, a copy of the program of a meeting of your Section held on Nov. 27, 1901, at which Dr. Hermann and myself read a paper on "Ulcero-Membranous Lesions of the Tonsils in Children, Associated with the Fusiform Bacillus (Vincent)."

On that same evening Dr. Emil Mayer read a paper entitled, "Affections of the Mouth and Throat in the Adult, Associated with the Fusiform Bacillus and Spirillum of Vincent, with Exhibition of Microscopic Specimens". Dr. Mayer's observations were based, if I remember rightly, on one or two cases, while our paper embraced a study of 12 cases in children. I also present to you a reprint of our original article, published in the *New York Medical Journal*, Dec. 7, 1901, Dr. Mayer's article appearing in the *American Journal of Medical Sciences*, February, 1902. Dr. Mayer unfortunately is unable to be here tonight, although you have heard his letter addressed to Dr. Albray. For that reason I have taken the floor to introduce myself as a sort of original Exhibit B in connection with this topic of Vincent's angina. The hour is too late to enter into the many phases of this subject, and I will content myself with some historical references. The paper by Dr. Hermann and myself—the first to appear in this country in the English language, and I believe in any other country—reminds me of the fate of a special tongue depressor for children which I invented some years ago. Two were sold, and I bought both of them. Our article, despite the fact that it was the first one published in this country on the subject, has never to my knowledge been referred to by the many American physicians who have written upon or discussed Vincent's angina and allied conditions. It has been left for two of us, Hermann and myself, to refer occasionally to our own work. Perhaps it will interest you to learn how Dr. Hermann and myself came to the study of Vincent's angina. At the time we were in charge of the Children's Clinic at the Good Samaritan Dispensary, and as one or more of these cases presented themselves, we looked upon them as clinical diphtheria and treated them as such. The cultures as forwarded to the Department of Health were invariably returned as negative for Klebs-Loeffler, one after the other. We became skeptical and checked up these cultures by making cultures in duplicate, sending one to the Department of Health and the other to our own laboratory. Our cultures were all negative, as were those of the Department of Health; and then, while we had more respect for the findings of the Health Department, we still were at a loss as to what the condition was which confronted us. We then decided to use smears in addition to cultures and from the very first a strange picture of fusiform bacilli and spirilla came into view. A study of the literature and our article followed. With it all, I believe that I can say with due modesty that not so very much new has been added to what was said by Dr. Hermann and myself in 1901, except, perhaps, for such studies of cultural growth as noted by Drs. Barker and Tenney, together with references to Tunnicliff, Krumwiede, Pilot and Davis and others, a wider appreciation of the fact that lesions similar to Vincent's angina occur on mucous membranes other than the mouth and on the skin, and the close study of ulcerative mouth lesions by the dental profession, no outstanding advance has been made. Dr. Barker spoke of the possibility of the fusiform bacillus and the spirillum being one and the same organism. In our original article, Dr. Hermann and I stated, "Notwithstanding the difference in size and shape, it is highly probable that the fusiform bacillus and the spirillum sputigenum are identical. Observations of transitional forms which we have made in our series of cases would seem to indicate that there is a genetic connection between the fusiform bacillus and the spirochete. Until all three conditions, as laid down by Koch—constant presence, pure cultures and experimental inoculation—are fulfilled, it cannot be stated with absolute certainty that the fusiform bacillus stands in a direct causative relation to this form of ulceromembranous angina. The following points, however, make its specific character highly probable: 1. Their uniform presence in very large numbers, or in nearly pure culture. 2. Their gradual disappearance during the process of healing. 3. The presence of so few other micro-organisms.

We also emphasized at that time the need of differentiation clinically from diphtheria and syphilis, our inability to secure pure cultures of the organism on

the then known media, the two clinical types—diphtheroid and ulceromembranous—the reliance for diagnosis upon smear rather than culture, the advisability in making the smear of entering deeply into the ulcerative ornecrotic mass, as well as many other points. I must agree, however, with Dr. Barker, that fusospirilosis or fusispirochetosis is a far better descriptive term of the condition under discussion. Dr. Tenney made a statement, "No teeth, no Vincent's. On this point I quote from Dr. Hermann's article on the Etiology of Noma. "That the teeth play an important part in the production of ulcerative and gangrenous stomatitis is shown by the following facts:

1. These processes are not met with where there are no teeth, namely, in infants and in old age.
2. The primary lesion is a gingivitis. The cheek, tongue, tonsils and other parts are always affected secondarily.
3. According to Kraus, in cases of ulcerative stomatitis, if a fine probe be introduced between the swollen inflamed gum and the tooth, the smallest, freshest ulcerations will be seen on the inner surface of the gum. From this point they spread to the edge and external surface.
4. In infants, in whom there are only two incisor teeth, the process begins adjacent to and opposite these teeth.
5. The greater frequency of ulcerative tomatitis at the time of the first and second dentition, which are often accompanied by hyperemia of the gums.
6. The organism associated with these processes is not found in lesions of the mouth of infants who have as yet no teeth.
7. The ulceration heals quickly when the offending tooth is removed.

From the therapeutic standpoint I should like to emphasize what has already been mentioned. The bacillus and spirillum grow and thrive anerobically. All successful cultures have been grown this way. The fundamental and basic principle in therapy is to bring oxygen to the affected areas, to produce conditions inimical to the growth of the organism. For that reason I believe that peroxid of hydrogen and perborate of soda either in saturated solution or in paste form are valuable. I have used arsenical preparations intramuscularly and locally with good results. A very useful formula, that of Paterson, I believe, consists of Liq. Potass. Arsenitiis, Vini Ipecac, Glycerini, Aq. Menth. Piper. Sig: 15 drops in two teaspoonsful of water and apply locally.

Naturally, as physicians we approach the subject from a somewhat different angle than dentists, and the technique of treatment as outlined by Dr. Albray appeals to me. Of course, there is no doubt that the fusiform bacillus and spirillum are normal inhabitants of the mouth. If you insert a platinum loop at the gingival margin of a slightly inflamed mouth, and make a smear stained with carbol fuchsin you will find some of these organisms. It is only when the mouth tissues undergo a physiological change, when conditions develop favorable for the growth of these organisms, such as we see during the course of infectious diseases, and blood or constitutional diseases, or in cases of unclean mouth that ulcerative lesions of Vincent develop.

I thank you, Mr. Chairman, for the courtesy of the floor, and I am glad to have come back here after 26 years to take part in the discussion of a subject which has interested me for so long a time.

DR. R. A. ALBRAY: I greatly appreciate the kindness with which you have received my paper. One of the gentlemen referred to the necessity for making Wassermann tests when treating Vincent's disease. You will recall that I stated in my paper that it is well to have a Wassermann test made, at least with patients where the disease is running a severe acute course, or did not respond readily to treatment.

Regarding the use of formaldehyde as a mouth wash, even up to 40 per cent solutions, I certainly would not care to have it used in my mouth. Perhaps some day I will try it for a patient, and if I am still alive after doing so will publish a report of the case. I have not seen the article which the Doctor quotes relative to salvarsan having no effect on the spirillum of Vincent in laboratory experimentation. I do know, however, that its use in the mouth accomplishes results which no other medicament has given in my practice and I will continue to use it until something better is discovered. Copper sulphate has given results second only to salvarsan.

Another speaker referred to a statement which had been made some time ago to the effect "no teeth, no angina". I stated in my paper that when there has been considerable destruction around and between the teeth and the organisms have located in these deeper structures, I did not believe they could ever be entirely eradicated until the teeth are all removed. Thank you.

DR. CREIGHTON BARKER: About once a year I take a Roman holiday and sit at one of these Vincent's infection round tables and hear a lot of valuable opinions and a few less facts. I wish I had been present when Dr. Sobel presented his argument 26 years ago; although much water has gone over the dam since, it has not produced much power. All of us have some method of therapy, and they all work. Someone has remarked in regard to just this subject, "Their use is founded on faith and that faith should be the guide to their use". I think he is correct. If you have faith in salvarsan, in copper sulphate, in vinegar, in lambs' urine, use that. But there are some facts that we want. If the gentleman will provide me with the identity of the article stating that salvarsan has no effect on these cultures, I will be much obliged to him. I don't promise to believe it, but I would like to read it. Let us go after facts in this matter, as we do for facts about everything else. It is all right to have your tricks of therapy, but if they are not founded on facts, to my mind they are not medicine.

DR. C. F. TENNEY: I wish to express my appreciation of Dr. Barker's paper, for he has done a splendid piece of work. We have all been fussing over Vincent's angina for a long time. Everyone has had a sort of fling at it, but it is by this persistent trying to do what we can, and presenting it, and having it torn to pieces that we may learn something. Dr. Barker is hammering away, and he is apt to get somewhere. He is a little warmer in the search than I have heard before.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, January 23, 1928.

Neurosis of the Nose. Dr. Wolff Freudenthal.
(*To appear in a subsequent issue of* THE LARYNGOSCOPE.)

Paper: The Diagnostic Barium Bougie and Lesions at the Lower End of the Esophagus. Dr. Harris P. Mosher (Boston).

DISCUSSION.

DR. H. H. FORBES: I wish to thank the Chairman of the Section for the opportunity he has given me to open the discussion of Dr. Mosher's paper, and I also wish to express my appreciation to Dr. Mosher for what he has given us this evening. It would, indeed, be an anticlimax for me to attempt to add or detract from the paper—in fact, either would be impossible. Dr. Mosher has made the lower end of the esophagus the subject of much study. When in Boston, a visit to the Harvard Medical School Anatomical Museum will amply repay any member of the Academy interested in esophagoscopy. There you will find anatomical and pathological specimens of the esophagus, as well as interesting models. The work following along the lines of Dr. Mosher, I was much pleased to note had been taken up by Dr. Braun Kelly, of Glasgow, and Mr. Hill, of London. All efforts to study the esophagus which add to the safety of the operation must be noted by us and passed along to the general practitioner. Our margin of safety has been increased wonderfully in the past ten years. I was discouraged in my early work about finding cases for examination, the internist feeling that it was too dangerous an operation to submit the patients to. Now there can be no excuse for not using the osophagoscope in all cases of unexplained dysphagia, both for diagnosis and, in many cases, for treatment—certainly in many cases described by Dr. Mosher and with the very useful instrument which he has devised.

Will Dr. Mosher, in closing the discussion, explain more fully the steps which he employs in treating a case of cardiospasm?

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

Meeting of Jan. 3, 1928.

COLLEGE OF PHYSICIANS.

DR. S. R. SKILLERN, JR., reported two cases of sinusitis with operation.

DISCUSSION.

DR. H. A. LAESSLE: Dr. Skillern's paper corroborates our work in the clinic. We have found that the needle punctures are sometimes very much more important than the X-ray findings. With reference to ethmoids, quite a number of X-ray reports have come back "Ethmoids Cloudy" and in these cases if we took a Hayek hook, put it under the middle turbinate and brought it forward, we would usually find pus. As regards the antrum conditions, I have in some instances washed the maxillary sinuses from 75 to 80 times, using various solutions, including dye preparations, and in practically all cases have gotten good results, with the exception when we find we have necrosed mucous membrane and destruction of bone, do we operate.

DR. HENRY S. WIEDER: What is the experience of the Skillern Clinic with transillumination as compared to X-rays? In my experience, transillumination is more reliable in maxillary conditions than X-ray.

I would also like to know what the attitude of the Skillern Clinic would be to a case such as was seen by me within the last few days. During a routine examination of the new patient, who complained of no headache, discharge or other symptoms of sinus disease, transillumination revealed a darkened antrum on one side. There was no pus in the nose on inspection nor on douching. Would this patient be punctured or would you wait for clinical symptoms of trouble before puncturing? Personally, I did not puncture as I have frequently punctured a darkened sinus, obtaining a dry tap, and I feel that there is always present the danger of infecting a sinus that was uninfected previous to puncture.

DR. PHILIP S. STOUT: In the Asthma Clinic at the Jefferson Hospital we find that X-ray is quite an advantage in our diagnosis of chronic sinus trouble. Of course, a sufficient number of pictures must be made to show all of the sinuses. Although transillumination is valuable, our experience is that it is not quite as conclusive as the X-ray. However, there was only 4 per cent variation between the X-ray and transillumination in the findings in a large series of bronchial asthma cases.

In connection with the diagnosis of antral conditions we use the antrascope, which is, as you know, an instrument for looking directly into the maxillary sinus through a perforation under the inferior turbinate.

DR. HARRY A. SCHATZ: I would like to know what the routine after-treatment is in Dr. Skillern's Clinic following radical antrum operations. Do they irrigate or not, and if so, with what? Also, how long does it take for the case to be entirely well? In my cases all treatment was completed at the end of 14 days after the operation. The solution I use is silver nitrate in suitable but ascending strengths. After having tried out, about a dozen years ago, the various agents then recommended, such as alcohol in ascending strengths, iodin in weak alcoholic or also in aqueous dilutions, Dichloramin-T in oily solution, and later, when it became popular, mercurochrome, all these proving unsatisfactory, I went back to the use of silver nitrate. Calculation led me to formulate the formula—3 drops of the stock 10 or 12 per cent solution dropped into 1 ounce of distilled water, the preliminary washing being, naturally, distilled water and not salt solution. Also, it is necessary to tilt the head so as to bring the solution in contact with all the surfaces within the antrum. At the next washing 4 drops are used, then 5, 6, 7, unless it shows rapid improvement, in which case the same strength is employed. Rarely can a patient tolerate more than 7 drops to the ounce without pain and burning in the side of the face. In unoperated cases if no improvement occurs with this

remedy, the recommendation of Dr. B. Alexander Randall is followed: aluminum acetate, 5 per cent suspension in water. This often rapidly cured cases that did not respond to silver.

DR. C. J. KISTLER: I would like to report my experience with the sinus cases that Dr. Skillern speaks about. My feeling and practice now is that in all upper sinus diseases of the nose where the antrum is also involved, the proper and permanent opening of the maxillary antra should be done first, because this will usually suffice to clear up the other conditions also. As regards the La Force instrument, I can corroborate what Dr. Skillern had to say concerning it. I have never been called to stop hemorrhage after the La Force operation, but I must say that I never use this instrument in adults—only in children up to, say, 15 years of age. After that age I prefer the snare and ligation.

DR. SAMUEL R. SKILLERN, JR. (closing discussion): Answering Dr. Wieder's questions: The valuation of transillumination as compared to the X-ray plate? We feel that more information is obtainable by transillumination at the time of our examination, than from an X-ray negative taken at some other time. Personally, I have sent a number of known purulent maxillary sinus cases to the Roentgenologist, with instructions to return for treatment as soon as the pictures were taken. I have then irrigated these sinuses and obtained up to an ounce of pus (some being of the thick, creamy variety). The following day the report and films would be sent me as negative, merely proving what our friends, the Roentgenologists, say, "Pus does not cast a shadow in all cases". The same abnormalities, such as thickened membrane, or bone, will affect both of these methods, and it is surprising the number of inequalities we find, while transilluminating the sinuses. Given a case of sinusitis, if we find one side darker than its fellow we needle and irrigate the maxillary, or if it be the frontal, and we cannot see any pus in the nose, after a thorough shrinkage of the Schneiderian membrane around the middle turbinate, then we refract that turbinate and watch for pus to appear under it.

When irrigating the maxillary sinus, do we accept a negative washing as a diagnosis that this sinus is not infected? We do not. It may be that the sinus has just recently emptied itself, or the pus may be so thick and tenacious, sticking so tightly to the floor of the sinus that the water will not dislodge it, or it may move around in the sinus and not get out through the ostium. If we suspect a maxillary of infection we irrigate it three or more times, at four-day intervals. Getting negative washings each time, we either rule this sinus out for the time being, or think of it as being abnormal in formation. In those cases in which the sinus has a high boney partition (or is divided into two sinuses, with a small opening in the partition), the irrigating fluid will not get into this pocket thus formed, and it may be that the mucous membrane of this pocket alone is infected. In these cases it is a good procedure to have the patient assume the prone posture, either lying on the side *not* affected, or turning the head so that the affected side will be the higher; this, of course, favors drainage toward the ostium. Upon resuming the examination there will either be found free pus in the nose or, more likely, pus in the washings from the needle irrigation. I have heard it said by general surgeons that pus is found the second or third washing, due to infection being carried in by the needle. This is not true if the Schneiderian membrane under and around the inferior turbinate is shrunken and deadened by cocain and the nose irrigated with sterile normal saline, before the needle puncture is made (with the needle taken directly from the sterilizer), why should this mucous membrane become infected and the nasal membrane remain clean? After the original needle puncture is made it is a painless procedure, with the aid of a little cocain, to reinsert the needle any number of times through this opening.

Now as to the antrascope for diagnosing. We do not use it. Personally, I have tried it a number of times but I have never been able to see pus in the antrum. The little window always gets fogged by either blood or some secretion and I cannot see through this fog. I have seen the inside of a few healthy antrums with it.

Packing the antrum after a radical operation: We do not believe this to be absolutely necessary, since we have been cutting away the mucous membrane under the inferior turbinate, but we continue to pack both the antrum and nose on the operated side to control any postoperative bleeding. The nasal

packing is removed in 24 hours, the sinus packing in 48 hours. Our after-treatment consists of irrigation with normal saline and the covering of the mucous membrane with a bland oil spray.

Herpes of the Soft Palate. Dr. James E. Landis.

(To appear in a subsequent issue of THE LARYNGOSCOPE.)

DISCUSSION.

DR. HARRY A. SCHATZ: Dr. Landis has gone over the subject with a great deal of detail, and the suggestions for general treatment should be noted. To crystallize the diagnostic features I would repeat that where we encounter the subjective symptoms that go with herpes, plus shallow ulcers of a characteristic grouping, and situated on the lingual surface of the soft palate near or adjacent to the uvula, it is a case of herpes.

Fibroma Arising from the Jugular Bulb and Invading the Middle Ear and External Auditory Canal. Dr. George W. Mackenzie.

(Published in full in this issue.)

DISCUSSION.

DR. MYRON A. ZACKS: About two years ago I presented a paper at this Society, the findings of which were quite similar to those presented in Dr. Mackenzie's case this evening. The diagnosis was in doubt for some time. My patient had been examined by various men and a multiplicity of opinions expressed. The original title of the paper was to have been "Unresolved Blood Clot in Middle Ear", but upon speaking to Dr. Mackenzie regarding this interesting case which we examined and studied together it was titled Hemosiderosis. As far as I was able to learn, after a careful survey of literature, it was the only case of its type on record at the time. There was nothing in the history of the patient of any aid concerning its etiology and it was discovered in the routine examination. There was evidently a striking similarity in the color of the drumhead in both cases. I distinctly recall the surprise when I witnessed for the first time this rare and most interesting condition and was tempted to incise the drum, but the possible danger of wounding an exposed jugular bulb prevented me from so doing. The X-ray report of Dr. Mackenzie's case is most interesting and may prove of valuable diagnostic aid when confronted with this condition.

Meeting of February 7, 1928.

COLLEGE OF PHYSICIANS.

1. Presentation of Case: "Carcinoma of the Sinuses in a Young Patient." Dr. H. A. Schatz.
2. Paper and Case Presentation: "Interesting Cochlear and Vestibular Findings Following Accident." Dr. M. A. Zacks.
3. Presentation of Patient: "Five Mastoidectomies and One Decompression On Same Patient." Dr. Philip S. Stout.
4. Paper: "Basilar Skull Fracture Involving the Left Temporal Bone." Dr. Wm. G. Shemeley.

Interesting Cochlear and Vestibular Findings Following Accident. Dr. Myron A. Zacks.

On Nov. 27, 1925, Case W. C., age 32 years, white, male, carpenter, married, was referred to the Department of Otology, Polyclinic Hospital, service of Dr. Walter Roberts, with the following history: Admitted to Frankford Hospital, Philadelphia, June 21, 1925, in an unconscious state. Unable to obtain any information because of patient's condition.

Present Illness: Immediately preceding admission to hospital, auto in which pationt was riding was struck by a trolley car and patient picked up on street in an unconscious condition and removed to the hospital.

Physical Examination: Adult male, about 32 years of age, lying in bed, unconscious, tossing from side to side, especially when touched. Vomiting frequently. Head and Neck: Laceration of scalp 3 to 4 inches long, left frontal region (sutured in Accident Ward). Eyes: Contusion of upper left eyelid. Pupils are equal and react sluggishly to light. Face: Multiple small

lacerations. Nose, Throat, Ears: No apparent injuries of any character. Chest: Normal shape, expansion slight, equal. Breath sounds normal. Sighing respiration. No rales. Heart: Rate 70, regular, no thrills, no murmurs, sounds are full and strong, heard best in midline, epigastric region. Abdomen: Soft, well relaxed; no apparent tenderness, no rigidity, no masses, no injuries. Extremities: Negative. No injuries. Reflexes: Knee jerks slightly sluggish. No Babinski.

Blood Pressure: 130/78.

June 22 ,1925: Patient vomiting frequently during day. Unconscious. Pupils react to light—equal.

X-ray Report: June 22, 1925: No X-ray evidence of fractured skull. No X-ray evidence of fracture of the pelvis. June 25: Eye examination: Left eye moderate neuroretinitis. Right eye negative.

X-ray Re-examination: June 25, 1025: The skull fails to show any X-ray evidence of fracture. June 28: Ear examination—Right ear: Drum moderately congested in posterior portion. Left ear: Severe congestion in posterior superior portion of external canal. Drum of normal color. Bone conduction taken over both mastoids is greatly impaired but not absent. June 30: Patient treated with sod. iodid. No disturbance of mentality. Patient is very deaf. July 4: Patient is clear mentally. No improvement in hearing. July 9: Laceration completely healed. Hearing not improved.

Discharged: After an 18-day stay in the hospital, the patient was discharged on July 9, 1925.

Nov. 27, 1925: Patient now, after an elapse of over five months, presents himself, complaining of total deafness, both ears, attacks of vertigo which are subsiding and for which he is compelled to use a cane. The staggering, which was at first pronounced, is now improving. At present there is no nausea or vomiting. The sense of taste and smell were lost for a period of several weeks following accident. There is no history of diplopia. At no time were the facial muscles involved. The mental makeup of the patient has not been altered at any time. Absence of any type of discharge from either ear prior to, during, or following accident. Patient states that hearing was perfect prior to accident, following which deafness has been complete.

Functional Hearing Tests: Nov. 27, 1925: Unable to appreciate sounds from any of the tuning forks or Galton whistle. When the 256 d. v. is caused to vibrate loudly and placed on the vertex, the vibration is felt but not heard. With the speaking tube applied to either ear, patient is unable to understand the whispered, spoken or shouted voice.

Wassermann (Blood): Dec. 12, 1925: Negative.

X-ray Report: Dec. 15, 1925: No evidence of pathology is present. We are unable to demonstrate a site of fracture, either old or recent. Sella turcica appears normal in size and outline. Sinuses appear normal.

Nose, Throat and Ear Examination: Ear: Mucous membrane of normal color. Septum thick, particularly high up. There is a marked ridge along the suture line on the right side impinging against the inferior turbinate, more or less occluding this side of the nose. There is a concavity of the septum on the left side with a compensatory hypertrophy of the left inferior turbinate. The right middle turbinate is jammed between the septum and nasolateral wall. The nose is free from abnormal secretion. Throat: The pharynx is slightly reddened. The tonsils are embedded and diseased. The lingual tonsil is moderately enlarged and contains a few superficial varicosities. Palatal muscles move freely and equally. Tongue protrudes in midline. Larynx: Negative for abnormal findings. Ears: Both tympanic membranes are brilliant, intact and show questionable retraction. There is no evidence, at this time, of injury to tympanic membrane or external canal of either side.

Vestibular Tests: Dec. 21, 1925: On looking to extreme right: Spontaneous nystagmus to right. On looking to extreme left: Spontaneous nystagmus to left. On looking up: Nystagmus absent. On looking down: Nystagmus absent. Romberg: Negative. Turning head to right: No effect. Turning head to left: No effect. Pelvic girdle movements: Good. There is no spontaneous past pointing.

Turning Tests: On turning patient to the right 10 times in 20 seconds, there is a horizontal nystagmus to the left of 11 seconds duration, of fair amplitude,

with vertigo of 13 seconds duration, with a past pointing of 3 inches to the right with right hand, and 2 inches to the right with the left hand. On turning patient to the left 10 times in 20 seconds, there is a horizontal nystagmus to the right of 11 seconds duration, of fair amplitude, with vertigo of 10 seconds duration, with a past pointing of 4 inches to the left with the right hand and touch with the left.

Caloric Tests: With the head lowered 30°, the right ear was douched with cold water (68° Fahrenheit) for the period of 4 minutes and no reaction observed except that after 2 minutes a questionable rotary nystagmus to the left occurred, very indefinite in character and immediately disappeared and did not recur during the next 2 minutes of douching. With the head back there was no response from the horizontal canal. Patient did not past point following douching of the vertical canals or horizontal canal.

On douching the left ear, with the head lowered 30°, with cold water (68° Fahrenheit) for a period of 4 minutes, there was no reaction obtained. Patient did not past point with either hand. With the head back, a horizontal nystagmus to the right of fair amplitude was obtained. The patient past pointed 4 inches to the left with the right hand and 4 inches to the left with the left hand.

Diagnosis: The cochlear function is entirely absent. The only canal which responds to stimulation is the left horizontal. While the responses to caloric stimulation resemble those found in cerebellopontile angle involvement, there is no involvement of either seventh nerve, hearing is absent in both ears, pelvic girdle movements are good and there is no spontaneous upward vertical nystagmus. It is difficult to explain these findings in view of the destruction of all elements except those found in the left horizontal canal which still react.

Ophthalmological Examination, Dr. Edwin B. Miller: O. D., 20/20. O. S., 20/20. O2. Cornea clear, anterior chambers normal. Pupils round, equal 3½ m.m., respond to light and accommodation. Ocular movements full. Convergence normal. O2. Media clear, discs somewhat oval. Margins well defined. Vessels slightly overfull. Macula and periphery negative. There is no ocular pathology.

Galvanic Test: May 4, 1926: Right side: Cathode, 7-9 ma., questionable rotary nystagmus to right. Anode, 3-4 ma., rotary nystagmus to left. Left side: Cathode, 6 ma., rotary nystagmus to left. Anode, 9 ma., rotary nystagmus to right.

Opinion: Eighth nerve on both sides reacts to galvanic stimulation.

Vestibular Test: Jan. 7, 1927: Findings similar to that of Dec. 11, 1925, except that there is an occasional upward spontaneous twitch upon looking upward, and the pelvic girdle movements are only fair. The response to caloric stimulation is as of previous date.

Audiogram: Dec. 9, 1927: Right ear: Total deafness to all sounds from C (64 d. v.) to c^6 (8192 d. v.). Left ear: Identical with findings noted in right except that with dial at approximately 85, the c^3 (512 d. v.) is definitely and unmistakably heard. This represents a 68 per cent hearing loss for this single remnant.

Diagnosis: Complete loss of cochlear function right ear. Cochlear function in left ear is completely gone except for c^3, of intensity of above mentioned.

Audiogram: Jan. 9, 1928: Identical with findings observed one month ago except that loss of hearing in left ear is 72 per cent instead of 68 per cent as previously noted.

Vestibular Test: Jan. 9, 1928: More or less identical with those previously noted.

Hearing Test: Feb. 2, 1928: With speaking tube in either ear, the whispered, spoken or shouted voice was not heard. The Weber test lateralized to the right, Schwabach taken over the vertex and mastoid bone is greatly shortened. Rinne is strongly negative. Patient thinks he hears the c^4 forks occasionally in the left ear. With Barany noise apparatus in right ear no sound is heard but when placed in left ear patient thinks he hears an occasional noise.

Stenger Test: Feb. 2, 1928: Findings constant on repeated examinations. Sound from vibrating fork absent from right ear. Occasionally heard in the left.

Caloric Test: Feb. 2, 1928: On douching the right ear with cold tap water 40-50° Fahrenheit) with head erect, no response is obtained from the vertical canals. With the head back, or head placed on the opposite shoulder, no response is obtained from the horizontal canal. On douching the left ear with cold tap water (40-50° Fahrenheit) with head erect, a rotary nystagmus to the right was obtained. With the head back, a good horizontal nystagmus to the right was noted. With the head placed on the right shoulder a good horizontal nystagmus to the left was noted. It is interesting to note that while cold water (68° Fahrenheit) on many previous and subsequent examinations failed to produce any response from the left vertical canals, much colder water (cold tap water, 40-50° Fahrenheit) produced a typical caloric response.

Galvanic Test: Feb. 2, 1928: Right ear: Cathode, 8-9 ma., rotary nystagmus to right. Anode, 5 ma., rotary nystagmus to left. Left ear: Cathode, 3 ma., rotary nystagmus to left. Anode, 7 ma., rotary nystagmus to right.

Comment: Inasmuch as the patient did not come under our care until five months following the accident, we are obviously unable to give the exact early clinical picture and pertinent findings, ipsofar as the labyrinthine disturbance is concerned. The hospital record, however, reveals an accident of such severity as to cause unconsciousness for three days, accompanied by frequent attacks of vomiting and symptoms of cerebral irritability. The hearing function which is alleged to have been perfect prior to the accident has been totally and persistently absent from the time consciousness was regained. Vertigo and staggering, which were severe immediately following the accident, subsided gradually with practically complete disappearance during the course of the next six or seven months. Extra ocular and facial muscles have never shown any involvement.

We were, therefore, at this late time confronted with the problem of locating and defining such lesion or lesions capable of explaining the present clinical findings, and which at the same time would harmonize with the early clinical picture.

A few of the many possibilities which suggested themselves are: 1. Fracture involving the petrous portion of one or both temporal bones, damaging or severing one or both eighth nerves. 2. Hemorrhage into one or both labyrinths. 3. Concussion of such degree as to injure or destroy the cochlear and vestibular filaments of one or both eighth nerves. 4. Damage in the region of the cerebellopontile angles or to the brain stem itself. 5. Concussion neurosis. 6. A combination, with simultaneous coexistence, of any of the above.

Further study was then undertaken, which disclosed findings of a most interesting and confusing type.

Our first problem was to determine whether we were dealing with a peripheral or central lesion or both. Space and the time limit prohibits mention of the differential factors except to remark that the weight of evidence exceedingly favored peripheral involvement. However, evidence of central involvement is not altogether lacking since the persistence of some hearing, though of tremendously limited quantity and quality, together with responses from the left horizontal canal, is significantly suggestive of its possible presence.

It is often most difficult, if not at times impossible, to detect a fissure fracture by X-ray examination and it is in this type of case that the functional tests of the cochlear and vestibular apparatus plays an important role.

A severe injury to the vestibular apparatus practically always involves the cochlear apparatus and usually results in total deafness on the affected side.

Destruction of the vestibular nerve gives negative galvanic responses on the affected side. In our case, the vestibular nerve of both sides reacts to electrical stimulation.

We must not overlook the possibility of a poorly functioning left labyrinth prior to accident and which was not made manifest until traumatic destruction of the right. Routine examination often reveals for the first time impaired or even absence of internal ear function.

The most common causes for total destruction of the labyrinth resulting from an accident are: 1. Fracture involving the labyrinth. 2. Hemorrhage into labyrinth. 3. Concussion sequela.

Repeated negative X-ray findings does not conclusively exclude possibility of fracture through the labyrinth. Fracture and hemorrhage in the vast majority

of cases totally and permanently destroy both cochlear and vestibular functions, while concussions may not be of the severity to entirely destroy both these functions but permit certain portions to escape. This may explain the findings observed on the left side.

The galvanic tests show that both eighth nerves receive and transmit the electrical stimulus. The pathological changes are therefore located in the end organ of both sides, complete destruction on the right, while the left has escaped to some extent.

The striking dissimilarity of labyrinthine findings which offer many explanations and deductions, presented to the writer sufficient interest to justify the presentation of the case and paper.

In conclusion I wish to express my grateful appreciation to my chief, Dr. Walter Roberts, for the privilege of presenting this case, as well as for the willing and helpful advice which was always mine for the mere asking.

DISCUSSION.

DR. LEWIS FISHER: The case presented by Dr. Zacks is of more than purely academic interest because the findings, namely, a totally nonfunctioning ear on the right side—cochleal and vestibular, with a loss of function from the vertical semicircular canals of the opposite side but fair responses from the horizontal canal, closely simulate the "phenomenon-complex" of an angle tumor. If this patient does have a mass in the right cerebellopontile angle, active surgical interference is, of course, to be considered. The interpretation of the findings, therefore, presents a real problem in differentiation between multiple lesions resulting from the injury, and tumor formation in the right cerebellopontile angle.

I had the opportunity of examining this patient a number of times and I felt with Dr. Zacks that since the patient's pelvic girdle movements are good and that he shows no other evidence of intracranial involvement and since the hearing on the opposite side is also affected to a considerable extent, that an angle lesion can be safely excluded. Although the findings would suggest a rather peculiar type of injury to both labyrinths, yet it is well to remember that concussion may produce all sorts of freakish phenomena, and it is within the realms of possibility that the blow which this patient received concussed the right labyrinth to an extent of utter demoralization and injured the left labyrinth less severely, permitting the horizontal canal elements to escape practically unscathed.

There is one other phase of this case which might be mentioned and that is the medico-legal aspect. This patient claims to have lost his hearing as a result of that injury and might be engaged in a suit for damages. He might be under suspicion as a malingerer as far as his deafness is concerned and the otologist might be called upon to determine that point. In this case the examiner is able to render a positive opinion. The total loss of all vestibular function on the right side would make it extremely improbable that the cochlea escaped. The vestibular responses are objective and cannot be affected in any manner by the patient, and a definite lack of all responses to stimulation indicates a definite loss of function. From this it is fair to infer that the cochlea —the less resistant organ—has almost certainly suffered the same fate.

THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The annual meeting of the American Academy of Ophthalmology and Otolaryngology will be held in St. Louis, Mo., October 15-19, inclusive. Hotel Statler has been selected as headquarters.

Dr. W. P. Wherry, Executive Secretary.

IN MEMORIAM

JAMES ELMORE LOGAN, M. D.

When Dr. James Elmore Logan, of Kansas City, Mo., passed away March 24, 1928, another of the American pioneers and a member of the "Old Guard" in Oto-Laryngology was lost to the profession.

James Elmore Logan was born in Nicholasville, Ky., Oct. 16, 1861. He studied medicine at the University of Kentucky and at the University of Missouri and received his M. D. degree at Bellevue Hospital Medical College, New York, in 1884, coming immediately to Kansas City where he began his practice. In 1887, he was married to Miss Helen Richards, of Leavenworth.

Dr. Logan was president of the old Missouri Medical College, in Kansas City, from 1902 to 1911; he took an active part in the civic

developments of Kansas City; was a staunch Democrat, a member of the Upper House of the old City Council, and served as a member of the Board of Park Commissioners.

Early in his professional career, Dr. Logan confined his professional activities to Oto-Laryngology and received the recognition of his colleagues for his sound scientific work, his genial personality and his fearless and independent expressions and professional opinions.

Dr. Logan was President of the American Laryngological Association in 1910; President of the American Laryngological, Rhinological and Otological Society in 1906; he was a Fellow of the American Otological Society, the American College of Surgeons and the American Medical Association and was a member of the Missouri State Medical Association; he was an active member of the Board of several of the representative Kansas City Hospitals, a charter member and ex-President of the Kansas City Country Club, a former President of the Kansas City Club and a member of the University Club.

His contributions to Oto-Laryngologic literature were not numerous but they were always scholarly, dignified and indicative of the broad vision and the humanitarian instincts of the man.

A genial companion, a loyal friend and a polished gentleman,—it was an honor to associate with Dr. Logan in all of his professional, social or personal interests. He served Oto-Laryngology well and faithfully and always contributed his efforts gracefully and efficiently.

His friends and colleagues mourn his loss sincerely and feel proud to have been identified with him.

We extend our heartfelt sympathies to his bereaved family.